EXPERIMENTS WITH DRUGS

Studies in the relation between Personality, Learning Theory and Drug Action

Edited by

H. J. EYSENCK, Ph. D.

PROFESSOR OF PSYCHOLOGY, UNIVERSITY OF LONDON
DIRECTOR, PSYCHOLOGICAL LABORATORIES,
INSTITUTE OF PSYCHIATRY
PSYCHOLOGIST,
MAUDSLEY AND BETHLEM ROYAL HOSPITALS

PERGAMON PRESS
OXFORD · LONDON · NEW YORK · PARIS
1963

PERGAMON PRESS LTD.
Headington Hill Hall, Oxford
4 & 5 Fitzroy Square, London W. 1.

PERGAMON PRESS INC.
122 East 55th Street, New York 22, N.Y.

GAUTHIER–VILLARS,
55 Quai des Grands-Augustins, Paris 6

PERGAMON PRESS G.m.b.H.
Kaiserstrasse 75, Frankfurt am Main

Library of Congress Card No. 61-11548

Set in Times New Roman 10/12 pt and
printed in Hungary by Franklin Printing House

LIST OF CONTRIBUTORS

S. AIBA, Ph. D.,
M. CHOPPY, Ph. D.,
G. S. CLARIDGE, Ph. D.,
C. G. COSTELLO, Ph. D.,
J. EASTERBROOK, B. Sc.,
H. J. EYSENCK, Ph. D.,
B. N. GOMEZ, M. A.,
R. N. GOOCH, M. D.,
R. N. HERRINGTON, M. D.,
H. C. HOLLAND, Ph. D.,
J. B. LURIA, M. A.,
R. LYNN, Ph. D.,
E. RODNIGHT, Ph. D.,
J. SYLVESTER, M. A.,

ACKNOWLEDGEMENTS

THIS book would not have been written, nor the work reported in it completed, without the support we have received from the Wallace Laboratories, and specifically their President, Dr. F. M. Berger. We have received additional support for various investigations from the U.S. Army, under contract DA–91–591–EUC–1458; and from the National Institute of Mental Health. To them, and to the many subjects who took part in these experiments, our sincerest thanks are due.

FOREWORD

THIS book is a report of a series of experiments, designed to test a theory regarding the behavioural effects of certain groups of drugs. These are the so-called "depressant" and "excitant" (or "stimulant") drugs, which may be represented by sodium amytal, alcohol and meprobamate on the one hand, and caffeine, Dexedrine and Meratran on the other. The theory contends that these drugs act in such a fashion as to change the level of activity of certain fundamental psychological mechanisms, which are derived from, and defined in terms of modern experimental psychology, particularly modern learning theory. In particular, it is suggested that depressant drugs increase inhibitory potentials and decrease excitatory ones, while stimulant drugs have the opposite effect. In view of the fact that the concepts of "excitation" and "inhibition" are operationally defined by means of certain clearly demarcated experiments, the more or less rigorous deductions from a general theoretical framework which constitute the hall-mark of the hypothetico-deductive method can easily be transformed into experimental tests of the theory in question. It is the purpose of this book to bring before the reader a number of such experiments, together with the theoretical rationale underlying their development. It not may be entirely erroneous to state that this mode of procedure has not usually been characteristic of modern psychopharmacology, which has relied to an exaggerated extent upon blind empiricism, and has abjured as almost sinful all dalliance with psychological theory (Trouton and Eysenck, 1960).

It is of course very likely, and may almost be taken as axiomatic, that these molar psychological concepts of "excitation" and "inhibition" will have recognizable physiological counterparts. If no such isomorphism could ever be demonstrated, even after lengthy and determined study, then the writer at least would feel very doubtful about the continued usefulness of the psychological concepts. At the present time no confident physiological identification can be undertaken, although certain suggestive speculations may occur to many readers familiar with the more recent studies of the ascending reticular formation. A special chapter has been devoted to a discussion of these issues, but it should be emphasized that interesting and promising as these links between psychology and physiology appear, they have not yet reached the stage where they can be regarded as anything but suggestive. For the time being, the psychological concepts must stand on their own feet, and prove their worth, or lack of it, by the time-honoured test of all theories: the prediction of facts previously unknown. It is by this test that the hypotheses put forward in this book should be judged; we believe that the ratio of successful predictions is sufficiently high to make further work along these lines distinctly worth while. It is our hope that this feeling will also be shared by psychologists in other departments, and that they will replicate our experiments, and extend them so as to bring into the net other, hitherto untested, predictions; in order to be widely acceptable, a theory should receive experimental support from a broader base than can be provided by a single department.

It has often been pointed out that scientific reasoning and argumentation resembles a net or web, rather than a chain; the notion of the "nomological network" recently advocated as providing a superior method of validation for psychological theories has found adumbrations among logicians and philosophers of science at least since the time of

Galileo – as will be seen by the quotation from Haller which opens this book. Three strands in this network which constitutes our theory have already been indicated; modern learning theory, psycho-pharmacology, and neuro-physiology. There is, however, a fourth strand which may require special mention because it has not hitherto played much part in the experimental study of drug effects, but which is quite central to our own approach to this problem; that is the concept of *personality*. It is our belief that the main dimensions of personality are related closely to the main physiological systems of the body, and if excitation and inhibition are indeed as fundamental conceptions as modern learning theory would have us believe, then it would seem to follow that there should be a corresponding dimension of personality the two parts of which would be characterized, respectively, by high excitatory and low inhibitory potential (the *introvert*), and by low excitatory and high inhibitory potential (the *extravert*). The theory anticipates, of course, continuous gradation between these extremes, but it does insist firmly on the close relation between personality (as indexed by behaviour) and excitation/inhibition balance (as indexed by laboratory test performance). Taking this hypothesis together with those already discussed, we see that it must follow that depressant drugs have an extraverting effect, while stimulant drugs have an introverting effect (Eysenck, 1957).

The relationships between these various concepts, all apparently lying at different levels of discourse, are discussed in the first chapter, which takes up the task of "setting the stage". It discusses the general way in which deductions can be made and predictions derived from the various parts of the underlying theory; it also shows some of the difficulties and dangers involved in too simple-minded and uncritical a reading of the theory. It summarizes many of the experimental studies which have preceded the present volume, and in which we have tested a wide variety of deductions. The remaining chapters each deal with a single, clearly demarcated subject and state the logic of the reasoning, as well as the general supporting evidence available for the various steps involved in the argument. Sometimes it has been necessary to perform several experiments before the drug experiment itself could be undertaken or interpreted; this usually happened when the chain of argument had in it a weak link which required additional work to be performed before we would be reasonably sure in our own minds that the drug experiment could with advantage be performed. This is one of the features which distinguish the hypothetico-deductive approach from the less theory-oriented type of work that is perhaps more common in the psycho-pharmacological field; much could be said about the relative advantages and disadvantages of these different approaches.

It remains to state that while the Editor was responsible for the general theory which was being tested in each of the studies here reported, and while he was always available to give advice and help when requested, nevertheless each chapter represents the independent work and thinking of the author, who alone is responsible for the deductions made and the conclusions drawn. In most cases the Editor would be in full agreement with what has been written; in some cases he would be able to see arguments both for and against the contributor's opinion; in one or two cases he would tend to disagree. It seemed much more desirable to leave each contributor complete freedom to present his own case, even at the risk of ending up with several unresolved differences (as in the case of the effects of drugs on apparent movement, where the views of Costello and Sylvester are by no means in full agreement!), than to try and enforce some monolithic uniformity by arbitrarily laying down canons of interpretation. The reader, of course, may disagree with both Contributor and Editor, and that is as it ought to be; the contribution made by a set of experiments such as those discussed here lies in the experimental findings, and these may easily fit

in with theories quite different from those which inspired the work. Theories in science are expendable; facts are not. Theories are useful in so fas as they lead to the discovery of facts; as J. J. Thomson once said: "A theory in science is a policy rather than a creed". We hope that the reader will agree with us that our policy has been a fruitful one; we have no wish to impose a creed upon him, or upon ourselves.

H. J. EYSENCK

Natura in reticulum sua genera connexit, non in catenam: homines non possunt nisi catenam sequi, cum non plura simul sermone exponere.

A. VON HALLER (1768)

Chapter 1

PERSONALITY AND DRUG EFFECTS

H. J. EYSENCK

As pointed out in the Foreword, the experiments described in this book are all concerned with the testing of deductions from a quite general theory, and consequently a thorough understanding of this theory is important if the empirical studies are to be properly evaluated. The more specific theorems and hypotheses underlying the work described in the following chapters are stated there, but it seemed necessary to discuss in some detail the more general propositions, as well as some of the unresolved problems and difficulties to which they give rise. This seemed particularly important because the uses and abuses of theory in scientific work are not always properly understood by workers in the fields of psychology and psychiatry; much useless discussion may be avoided by clearly stating at the outset just what our contentions are.

The first point to be made in an outline of our theory concerns the postulation of two processes, entitled "inhibition" and "excitation". The first writer to use these terms and concepts in roughly the way here intended was of course Pavlov (1927); among those who have clarified their meaning and added experimental content Hull (1943) must be particularly singled out. Many other writers, from Dodge (1928; 1931) to Teplov (1959), have contributed to the gradual clarification of these notions, but it would not be correct to say that any agreed and unambiguous definition of these two terms would be possible even now. To expect such a definition at this moment would indeed be to misunderstand the way in which science works and progresses. As Louis Pasteur has put it in his classic *Études sur la Bière*, "Science is built up by successive solutions given to questions of ever-increasing subtlety, approaching nearer and nearer towards the very essence of phenomena." Advance is therefore gradual, and perfection is not likely to characterize early or even intermediate stages of development. It is one of the aims of this book to help in the slow and arduous process which may ultimately lead to the proper understanding of the concepts of "excitation" and "inhibition"; none of the contributors would imagine for a single instance that such understanding had already been achieved.

Such an avowal of ignorance does not, however, imply that the use of these terms, and the theoretical developments outlined below, are of little or no value in guiding experimentation. Theories stand near the beginning of the long road that leads to scientific laws, and their main value is that of being guide-posts for the discovery of new facts which in turn will enable better theories to be built. Just as bad maps are better than no map at all, so even a poor theory may be useful in leading to a better understanding of a given field of knowledge, through leading to more thorough

exploration of the least well known parts, or those where discrepancies are discovered between theory and fact. Near the end of the road, where maps and theories are well-nigh perfect, they do not serve the purposes of exploration any longer, because there is no further need for exploration; theories have become laws, and serve only as memory aids summarizing experimental results.

Granted, then, that we cannot define "inhibition", we can at least say what kind of phenomenon is responsible for the postulation of a concept of this kind, and we can give a rough-and-ready statement of the sort of thing we shall intend by the use of the term. In its broadest meaning, then, inhibition refers to a process within the C.N.S. which interferes with the ongoing perceptual, cognitive and motor activities of the organism. The type of interference intended can best be clarified by reference to the two main types of inhibition, which may be called *temporal inhibition* and *spatial inhibition* (Eysenck, 1957). Temporal inhibition refers to the accumulation of a performance decrement as the result of the performance itself; it is usually associated with massed practice, and can be elicited experimentally in those situations giving rise to what Pavlov has called "internal inhibition" and Hull "reactive inhibition". Spatial inhibition refers to the production of a performance decrement through some other form of action occuring simultaneously, or almost simultaneously; it is sometimes called "distraction" in common parlance, and is similar to Pavlov's notion of "external inhibition". The terminology here suggested is preferred to that of Pavlov or Hull because these authors have given to their own terms excess meaning by incorporating them in a wider theoretical system, so that the use of Hullian or Pavlovian terms might seem to imply acceptance of that system as a whole*. Much as we admire the work of both scientists, we cannot believe that at the present time the unchanged use of their theories could properly be defended, so that more neutral terms seemed preferable.

We may now turn to the experimental paradigms which could be used to define operationally the concepts of temporal and spatial inhibition. Only a few of these will be mentioned here as no complete account of the general theory could possibly be given within the confines of this chapter. Consider then as one example the facts relating to the well-known phenomenon of *reminiscence* (McGeoch and Irion, 1952). If a motor or perceptual task is carried out by the subject under conditions of massed practice, i.e. without or with minimal rest pauses, then the theory demands that he should develop temporal inhibition; this inhibition, being a fatigue-like state, should interfere with performance, and should dissipate during rest after the termination of the scheduled performance. If, then, the subject were asked to resume practice after the rest pause, then his per-

* The objections to Pavlov's physiological notions are too well known to require restatement here (Konorski, 1940). Hull's concept of reactive inhibition is peripheralist and tied to the idea of actual physical work done by the organism; neither of these views is tenable any longer (Eysenck, 1957; Broadbent, 1958). Our formulation escapes many of the strictures which Broadbent has levelled against the Hullian theory; his own "filter" type of hypothesis to account for the facts of inhibition, while plausible when perceptual data alone are considered, breaks down when we attempt to explain reminiscence and other similar phenomena.

formance should appear to have *improved* when a comparison is made of his scores immediately before and immediately after the rest pause. This improvement, which has often been demonstrated experimentally, is technically known as reminiscence, and in spite of many efforts to account for this phenomenon along different lines there is considerable agreement among experimental psychologists that it can be most readily understood in terms of temporal inhibition.

Another phenomenon which would appear to demand a similar explanation is that of *vigilance*, or rather the decline of vigilance. Whenever a long-continued and monotonous visual or auditory task is being carried out, such as the continued inspection of a series of dials one of which may occasionally indicate danger, or the continued listening to a series of numbers one of which may occasionally require some action to be performed, then it is usually found that performance is perfect or near-perfect at the beginning, but declines after a while to an altogether lower level. This decline in "vigilance" may be supposed to be due to temporal inhibition, and the recovery after rest may be considered analogous to "reminiscence".

A third phenomenon which may be relevant is that of adaptation. Whenever a stimulus is presented to an organism, certain non-specific reactions occur such as the orienting reflex (Berlyne, 1960; Razran, 1961), changes in the electric conductivity of the skin (Martin, 1960), and various other autonomic reactions. On repetition, particularly on rapid (massed) repetition of the stimulus, these reactions grow less strong and may in time die out completely. While peripheral events may play some part in this process of adaptation, there is no doubt that central factors are involved also, and it is the involvement of these which may make this series of phenomena relevant to the notion of temporal inhibition.

With respect to spatial inhibition, we may perhaps refer to the well-known process called "extinction" by Bender (1952), which he defines as "a process in which the sensation disappears or a stimulus becomes imperceptible when another sensation is evoked by simultaneous stimulation elsewhere in the sensory field". Bender's own work, and that of the authors quoted by him, is rather poorly controlled and of an all-or-none character, but the more recent studies of Uttal (1961) and Ingham (1959) show that corresponding phenomena can be obtained in well-controlled and properly quantified experiments. Further discussion of this type of inhibition, as well as a somewhat extended list of experiments demonstrating both temporal and spatial inhibition, has been given on an earlier occasion (Eysenck, 1957), and the present examples are mentioned only to give the reader unfamiliar with experimental psychology an opportunity of discovering the empirical background of the terms used.

We have restricted our discussion of cortical inhibition to *stimulus-produced* inhibition. Klein and Krech (1952) have argued in favour of the view that attention should also be paid to the general state of excitation or inhibition of the cortex; they say: "We would postulate another factor which contributes to the extent of drop in cortical conductivity: we would assume that the *over-all state of the cortex* helps to determine the initial or *basal* value of cortical conductivity and the degree of drop *possible*.

For example, individuals may be thought of as having high or low cortical conductivity *prior to any stimulation*, i.e. the basal or characteristic level of cortical conductivity." As Eysenck (1957) has argued, this proposal in its original form is not capable of proper experimental investigation. It may, nevertheless, contain some important elements of truth.

In the first place, it is possible that the constant stream of sensory and proprioceptive bombardment to which we are exposed all day sets up inhibitions of the stimulus-produced kind which has no time to dissipate completely; this would be particularly true of "stimulus-satiation" as contrasted with "reactive inhibition" in view of the demonstrable longer persistence of the former (Thompson, 1960). In view of the markedly slower dissipation of stimulus-produced inhibition in schizophrenics (Eysenck, 1961), we would expect this factor to produce marked differences in schizophrenics tested early in the morning and later in the day; this has been done successfully by Streltsova (1955).

In the second place, it is possible to imagine a constitutional property of the central nervous system such that it is constantly (at least during waking hours) in a state of greater of lesser excitation or inhibition. One could then think of drugs as altering this central state, thus temporarily changing this "constitutional" property. One might in this connexion think of such concepts as "arousal" (Berlyne, 1960) although of course there are considerable difficulties in designing any test of the differential existence of such a "state" without introducing some kind of stimulation which would immediately confound any "state"-effects with stimulus-produced effects. If the only experimentally demonstrable effect of the "state" is to increase or decrease stimulus-produced effects, then we are justified in concentrating on experimental investigations involving the latter alone.

On the purely theoretical level, of course, "cortical conductivity" is an unavoidable construct if we postulate consistent individual differences in stimulus-produced inhibition.

It is entirely reasonable from the physiological point of view to suppose that the excitatory state of a system, or group of cells, will somehow affect the passage of a nerve impulse through it, and there is indeed ample evidence (though of a scattered kind) that the form of change in electrical activity of the cortex in response to significant afferent stimuli depends upon the excitatory state of the cortex at the time of arrival of the stimulus (Jasper, 1938.) If we take seriously the suggestion that "arousal", as mediated by the reticular formation, may have some special connexion with the concept of "excitation", then it would be possible to obtain measures of this "state" without the imposition of extraneous stimuli; measures of electric conductivity, of E.E.G. patterning, and of many other autonomic and muscular (EMG) recordings in the so-called "resting state" may serve this function. Martin (1960) has reported an interesting experiment showing the dependence of GSR conditioning upon "arousal", and her comments, although specific to the conditioning process, may have wider implications: "The activity level of the system being conditioned seems . . . to play some part in the conditioning process, although this part may be more or less important in different modalities. *It is a factor which has been undeservedly*

neglected, particularly in theories which are concerned with individual differences." The implications are clear: if levels of autonomic and skeletal muscle activity can used to measure a central state of "excitation", then predictions can be made about the behaviour of individuals in a great variety of situations.

We must now turn to the definition of the concept of "excitation" (arousal?). In its broadest meaning, this term refers to a process within the C.N.S. which facilitates the ongoing perceptual, cognitive and motor activities of the organism. One particularly important meaning may relate to the notion of $_{S}H_{R}$ or habit-formation; in part at least this may be surmised to be a function of the organism's habitual or specific state of C.N.S excitation. Sensory thresholds, too, may be influenced to some extent by the organism's degree of excitation, in addition to the obvious peripheral factors which are probably of much greater importance in the determination of such thresholds; there is some evidence from the work of Teplov (1956; 1959) that this may be so, and that sensory thresholds may be correlated in predictable ways with personality. But these are on the whole rather speculative excursions into relatively unknown territory; they are of a much less certain standing than the well-substantiated facts of reminiscence or vigilance. The reason for this lack of evidence is closely connected with the obvious difficulties which must attend the postulation of two antagonistic processes, such as excitation and inhibition; any particular experimental fact can often be accounted for just as easily by invoking an excess of inhibition as by invoking a deficit of excitation and it must become a difficult and complex task to sort out the relative contribution and the degree of independence of these two processes. We shall return to this problem later.

For the moment, let us turn to the possible relevance of these processes to the study of personality. The writer has posulated that there is a close relationship between the personality dimension of extraversion – introversion and excitation/inhibition, in the sense that high degrees of extraversion are found in people in whom inhibitory processes occur quickly, strongly, and persistently, while excitatory processes occur slowly, weakly, and non-persistently; high degrees of introversion are found in people in whom the reverse is true. The concept of the introversion-extraversion dimension refers to a behavioural continuum on which each person's position can be ascertained with reasonable precision, either by obtaining ratings of his customary behaviour from persons qualified to judge it, or by questionnaires (self-ratings) when it can reasonably be assumed that the subject is not strongly motivated to give a distorted picture of himself (Eysenck, 1960a). According to the considerable amount of research done in this field since the pioneering studies of Heymans and Wiersma (cf. reference above), it appears that the typical extravert is sociable, likes parties, has many friends, needs to have people to talk to, and does not like reading or studying by himself. He craves excitement, takes chances, often sticks his neck out, acts on the spur of the moment and is generally an impulsive individual. He is fond of practical jokes, always has a ready answer and generally likes change; he is carefree, easygoing optimistic and likes

to "laugh and be merry". He prefers to keep moving and doing things, tends to be aggressive and loses his temper quickly; altogether his feelings are not kept under tight control and he is not always a reliable person.

The typical introvert is a quiet, retiring sort of person, introspective and fond of books rather than people; he is reserved and distant except to intimate friends. He tends to plan ahead, "looks before he leaps" and distrusts the impulse of the moment. He does not like excitement, takes matters of everyday life with proper seriousness and prefers a well-ordered mode of life. He keeps his feelings under close control, seldom behaves in an aggressive manner and does not lose his temper easily. He is reliable, somewhat pessimistic and places great value on ethical standards. (These pictures may look rather like caricatures because they describe as it were the *pure* or *ideal* type; living people of course only approximate more or less closely to one or the other extreme; they are not likely to resemble it in every detail.)

The original studies on this typology were performed by various European writers, among whom Heymans, Wiersma and Brugman stand out as the most sophisticated. They pioneered the use of statistical methods for the isolation of personality traits and were among the first to use rating methods; they even put forward a general theory to account for the personality dimension which they had isolated and performed laboratory experiments to test deductions from this theory. While their theory leans heavily on the Austrian psychiatrist Gross (1902; 1909) there is no doubt that these Dutch scientists have made a major contribution to the psychology of personality; the story has been traced in some detail in the writer's *Structure of Human Personality* (Eysenck, 1960a). The fact that Jung (1921), who added little to their work but suggest the use of the terms "extraversion" and "introversion" instead of the rather clumsy nomenclature used by them, is universally regarded as the creator of the whole typology is a sad comment on the lack of scholarship which is unfortunately characteristic of the whole field of personality study. (It is not widely known either that the terms "extraversion" and "introversion" themselves have been in use for several hundred years, and were not coined by Jung.) The one truly original contribution made by Jung, and it is not an unimportant one, was to suggest that this typology corresponded with another one which had been proposed by the great psychiatrist Janet in his descriptive and nosological work on the neuroses, i. e. the grouping of neurotics into two main clusters which he labelled *hysterics* and *psychasthenics*. The indentification of the hysterics with the extraverted end of the typological scheme, and of the psychasthenics with the introverted end, has been shown in later experimental work to have been fully justified (Eysenck, 1947; 1960b); it would appear now that the category of *psychopaths* is characterized by even more extreme extraversion than are the hysterics, and it would seem desirable to supplant the term *psychasthenia*, which is obsolete, by the more descriptive label *dysthymia* (Eysenck, 1952). But there is little doubt that on the whole the Janet-Jung hypothesis has been a fruitful and important one, and has led to much empirical work in the hands of clinical and experimental psychologists. It is interesting to note that the very extensive series of studies carried out independently by Cattell has led him to conclusions identical

with ours as far as the importance of the two dimensions of neuroticism and extraversion are concerned; he also finds an identical grouping of neurotic categories to ours in relation to these two factors (Cattell and Scheier, 1961).

Jung himself did not propose any causal theory of extraversion and introversion, although he seems to have leant towards environmentalistic explanations; Gross, and Heymans and Wiersma, however, did advance such an hypothesis in terms which appeared to be almost physiological. Briefly, they proposed that each primary mental act is followed by a secondary after-effect, a kind of perseverative effect, the duration of which is determined by the strength of the primary effect. Introverts have strong primary functions and correspondingly long secondary ones; extraverts have weak primary functions and short secondary ones. In this way are explained the changeableness, unreliability, impulsiveness and liking for excitement of the extravert, and his tendency to act on the spur of the moment; the preference of the introvert for planning ahead, his steadiness and reliability, and his liking for a well-ordered mode of life can equally easily be deduced from this theory. Its weakness lies in the pseudo-physiological character of the concepts, which do not refer to notions familiar to neurophysiologists, and the lack of experimentally testable deductions. The development of tests of "perseveration" by the Dutch school, and later on by Spearman (1927), might have obviated the second criticism, had it not been found that the search for such tests led into a quagmire rather than on to firm foundations for further advance (Eysenck, 1960a). The truth would appear to be that experimental psychology had not yet advanced sufficiently to provide these early workers with concepts securely founded in the laboratory, and relevant to their theory; their efforts to supply such concepts themselves could not be entirely successful under the circumstances.

In 1957 the writer suggested that experimental psychology was now ready to supply the missing link, and postulated that extraverts were characterized by strong inhibition and weak excitation, while introverts showed the opposite tendency (Eysenck, 1957). A good deal of experimental material was presented in support of this hypothesis, although most of the experimental studies cited dealt with temporal inhibition in one or other of its manifestations; little attention was paid to spatial inhibition, and even less to excitation. Further evidence was presented a few years later (Eysenck, 1960b), again concentrating very much on temporal inhibition; the writer feels that by now the available evidence may be said to support with some force the proposition that extraverts are characterized by quickly developing and slowly disappearing temporal (reactive) inhibition. Whether the extension of the theory to spatial inhibition and to excitation can be justified must be left to future experimental work; the results of such experiments, several of which are in progress at the present time, cannot of course affect the truth of the theory as far as temporal inhibition is concerned. It should also be noted here that certain further extensions of the theory have taken place in the sense that heightened inhibition has also been causally linked with: (a) brain operations and injuries, particularly prefrontal lobectomy (Petrie, 1952; Willett, 1960; Eysenck, 1957); (b) old

age (Eysenck, 1960c; Griew and Lynn, 1960); and (c) the administration of depressant drugs (Eysenck, 1957; Trouton and Eysenck, 1960). It is the third of these extensions, of course, that the present volume is concerned with.

How are these general postulates translated into experimental demonstrations, and how, precisely, are the deductions from the general theory related to the testing of these particular hypotheses? An example will make this vitally important point clearer than an abstract discussion. Consider Fig. 1. Along the abscissa we have plotted degrees of sensory stimulation, from extremely low at the left to extremely high on the right. Along the ordinate we have plotted the hedonic tone associated with these different

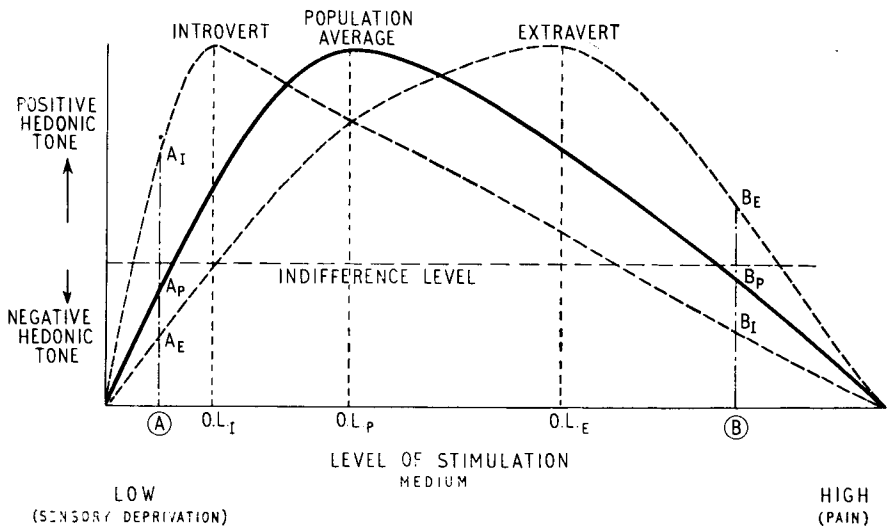


FIG. 1. Diagram representing relation between Level of Stimulation (abscissa) and Hedonic Tone (ordinate) in Extraverts and Introverts. For explanation see text.

levels of stimulation, ranging from strongly negative (feelings of displeasure or even pain; desire to escape, to end the stimulation, abience) to strongly positive (feelings of intense pleasure; desire to prolong the stimulation, or even to increase it, adience). Between the positive and negative hedonic tone there is an *indifference level*, indicating that stimulation is neither sought nor avoided, but is quite neutral to the subject. These concepts are discussed in technical detail by such writers as Beebee-Center (1932) and P.T. Young (1936. 1961), but for our present purposes we may accept them in a rather common-sense manner.

The strongly drawn curvilinear line in the centre of the diagram indicates the relationship between hedonic tone and strength of sensory stimulation, as derived from random samples of the population. We find that extremely high levels of stimulation produce pain and discomfort, and have consequently a high negative hedonic tone (Beecher, 1959). Extremely low levels of stimulation (sensory deprivation) have also been found to be productive of high negative hedonic tone and to be bearable only for relatively short periods (Beech, 1961). It is only at intermediate levels of sensory stimulation that positive hedonic tone develops, and this finding is not perhaps entirely out of line with common experience and expectation. In any case, there is ample experimental evidence in the literature cited for the general correctness of the picture presented in Fig. 1. For a detailed account of the relevant evidence see Berlyne (1960).* The curvilinear relation between tone and stimulus intensity was already postulated by Wundt (1874), although in an incomplete form.

Experimental psychologists often leave off when a general proposition such as the above has been enunciated, and feel that their work has been done, and that nothing further is required. The demonstration of a functional relationship between two variables, with everything else held constant, is deemed to be the *alpha* and *omega* of scientific endeavour; the very large individual differences which nearly always characterize the results are dismissed with a shrug of the shoulder as inevitable "random errors". The writer has suggested that such an attitude is quite unwarranted because the "errors" are not in fact "errors" at all, and are certainly not random; they form a necessary part of the study of the behaviour of organisms which differ from each other by heredity and by environmental background, and are susceptible to scientific research and enquiry (Eysenck, 1957). Let us consider in this particular case what kind of prediction our theory, linking extraversion and inhibition, would enable us to make.

If an individual is exposed to continued stimulation of a particular kind, then according to general experimental psychology adaptation/inhibition should set in, thus reducing the effective amount of stimulation received

* Berlyne (1960) points out that "it is tempting to suppose that the conditions that make for boredom will produce exceptionally low arousal, and that low arousal, as well as high arousal, must therefore be aversive". Hebb (1955) and others have indeed argued in favour of such a hypothesis, but we agree with Berlyne that the experimental data do not allow us to make any such identification; sensory deprivation seems to make for high, rather than for low arousal. We would also agree with his hypothesis that "the arousal tonus ... will require a particular rate of influx of arousal potential to maintain it. If the influx of arousal potential either exceeds or falls short of this rate, arousal will rise above the tonus level, increasing drive. Then a return to the tonus level will be rewarding, and any responses that promote such a return, whether by rectifying the excess or deficit of arousal potential or by compensating for it, will thus be reinforced and likely to recur in such circumstances". He goes on to say: "Our hypotheses imply, therefore, that, for an individual organism at a particular time, there will be an *optimal influx of arousal potential*.... The organism will thus strive to keep arousal potential near its optimum, which will normally be some distance from both the upper and the lower extreme." Other writers have approached a similar point of view, which may therefore be said to be rather widely acceptable (Dember and Earl, 1957; Glanzer, 1958; Hebb, 1955; Leuba, 1955; McClelland *et al.*, 1953; McReynolds, 1956.)

by the subject.* If it be true that extraverts show greater inhibition than introverts, with the "homme moyen" in between of course, then it would seem to follow that any given degree of stimulation would effectively be experienced by introverts as *higher* than it was being experienced by the average person, while similarly it would be experienced by extraverts as *lower* than it was being experienced by the average person. Objectively equal amounts of stimulation, therefore would not be experienced as equal by extraverts and introverts; they would appear displaced to the right of the abscissa of Fig. 1 by the introvert, and to the left by the extravert. Similarly, if O.L. represents the optimum (or preferred) level of stimulation of a given person, then $O.L._I$ would lie to the left of $O.L._P$, and this in turn to the left of $O.L._E$, where I and E refer to introvert and extravert, respectively, and P to the population average.

Again, consider two points, A and B, on the abscissa, referring to low and high stimulation respectively. If straight lines are drawn through these points, parallel to the ordinate, they will cross the general curve relating level of stimulation to hedonic tone roughly at the indifference level; in other words, for the avarge person these two stimuli are equally indifferent. For the typical extravert and introvert, however, as already explained, the general curve is not representative, and has to be displaced, to the left for the introvert, and to the right for the extravert. It follows, as shown in the diagram, that stimulus A will be positively hedonic for the introvert (A_I) and negatively hedonic for the extravert (A_E), while B will be negatively hedonic for the introvert (B_I) and positively hedonic for the extravert (B_E). (Similar consequences would appear to follow if we based our argument on individual differences in "excitation" rather than in "inhibition"; we are not concerned at this point with the possibility of a crucial experiment to decide between these alternative hypotheses.)

Many testable deductions follow from this hypothesis. Consider first the respective reactions of introverted and extraverted subjects to a test of *pain tolerance*, i.e. a test in which the subject is exposed to strong stimulation, and in which his score is the length of time elapsing from the beginning of the stimulation until he voluntarily withdraws, being unable to bear the pain any longer. It follows directly from Fig. 1 that with *identical* objective stimulation extraverts would experience less pain than introverts, due to the intervention of strong inhibitory potentials, and would therefore be more likely to tolerate the pain for long periods of time. Hence the prediction would seem to follow directly from our theory that *extraverts show greater pain tolerance than introverts.* Using different types of subjects, and also different pain-producing stimuli, Petrie (1960), Poser (1960), and Lynn and Eysenck (1961) have all reported significant positive relationships between extraversion and pain tolerance; the correlations in the two last-named studies were ·53 and ·69 respectively, using unselected groups. The evidence seems to support the prediction.

Consider next the reaction of extraverts and introverts to a test of *stimulus*

* The inhibition discussed here is of course conceived to be *central* in nature; peripheral processes of adaption are not here considered.

deprivation, i.e. a test in which the subject is exposed to as complete a deprivation of stimulation as can be arranged by the experimenter, and in which his score is the length of time elapsing from the beginning of the deprivation until he voluntarily withdraws, being unable to stand the deprivation any longer. It follows directly from Fig. 1 that with identical objective deprivation conditions extraverts would experience less stimulation (i.e. greater deprivation) than introverts, due to the intervention of strong inhibitory potentials, and would therefore be less likely to tolerate the deprivation for long periods of time. Hence the prediction would seem to follow directly from our theory that *extraverts show less stimulus deprivation tolerance than introverts.* This prediction also has received experimental support in the work of Petrie, Collins, and Solomon (1960). The complementary nature of the predictions, and their experimental verification, are important because many otherwise tempting alternative explanations are automatically ruled out, such as for instance an appeal to the greater motivation of one group or the other.

A third type of prediction follows from the different positions of the O.L. s (optimum levels of stimulation) of extraverts and introverts respectively on the abscissa in Fig. 1. We would deduce from this difference in position the existence of a kind of *stimulus hunger* on the part of the extraverts, and a *stimulus avoidance* on the part of the introverts, compared to each other. We would therefore predict that extraverts would be more likely to smoke more, drink more, and eat more (particularly spicy food) have intercourse more frequently, take more risks (with the accompanying autonomic stimulation providing what Berlyne (1960) has called an "arousal jag") and enjoy parties and social intercourse generally, because of the considerable stimulation provided. The evidence on these points is only partial; extraverts drink more (Eysenck, 1958) and smoke more cigarettes (Eysenck *et al.*, 1960), but are less addicted to pipe smoking than introverts; they have more illegitimate children (S.B.G. Eysenck, 1961), take more risks (Lynn, 1962) and are certainly more sociable (Eysenck, 1959). They also make larger movements (Rachman, 1961a), thus producing greater proprioceptive stimulation (Borg and Dahlström, 1960). (Rachman (1961b) also verified the corollary proposition that a stimulant drug would reduce the amount of motor movement.)

A fourth type of prediction relates to the assumption that perceptual after-effects, such as the length of visual after-images, and the length of the rotating spiral after-effect, are related to the amount (strength and/or length) of stimulation received; it follows from our theory that under conditions of equal stimulation extraverts would in fact, because of their strong inhibitory potentials, receive less effective stimulation. The prediction would therefore be that *extraverts show shorter perceptual after-effects.* (This prediction is supported by another consideration. The after-effect itself must be mediated in all likelihood by physiological processes which themselves are subject to inhibition; this inhibition being presumably stronger in extraverts than in introverts would lead to the same result. Both processes may be presumed to be active, and to support each other.) The evidence for these predictions, which is quite strong, is discussed in later chapters, and will not be summarized here.

Rather different, and a little more complex, is a fifth type of deduction. The writer has postulated the existence of another important personality dimension, additional to extraversion – introversion, to which the name "neuroticism" or "emotionality" has been given (Eysenck, 1947; 1960a). This is conceived of as being closely connected with an individual's autonomic lability, in such a way that subjects high on this factor show strong and long-continued autonomic reactions to standard stress tests, while those low on this factor show weak and short reactions. Neuroticism and extraversion – introversion were considered as quite separate and independent (orthogonal). It was however discovered that while this independence was indeed found to hold in normal populations, it ceased to apply in neurotic populations, or even in normal sub-groups having high scores on neuroticism (Eysenck, 1959). The explanation would seem to be that strong autonomic reactions are perceived as stimuli by the organism and are subject to temporal inhibition just as much as are other exteroceptive and proprioceptive stimuli; extraverts would consequently inhibit these stimuli more successfully than introverts, so that at high levels of neuroticism (strong and long-continued stimulation) extraverts originally generating just as much stimulation as introverts would soon drop to a lower level of stimulation, due to inhibition. This would lead directly to the observed interaction effect at high levels of neuroticism. Evidence for this hypothesis has been given elsewhere (Eysenck and Claridge, 1962).

A sixth and last deduction leads us into the field of aesthetics. There is some evidence that there are consistent individual differences regarding preferences for highly coloured as opposed to less highly coloured pictures (Eysenck, 1941), and it would seem to follow from our hypothesis that extraverts would prefer the highly coloured, introverts the less highy coloured ones. In some unpublished recent studies, the writer found quite highly significant correlations in the predicted direction, thus showing that in this field too fruitful deductions could be made from the general theory. (CF. Lynn and Butler, 1962).

It would be possible to continue listing predictions of this type, but there would appear little point in doing so; our main purpose was to illustrate the way in which deductions of an experimentally testable type are generated within the framework of the theory under discussion. Reference has preciously been made of the phenomena of *reminiscence* and *vigilance* as characteristic of temporal inhibition; our general theory would predict that these also should show predictable relationships with extraversion, and indeed it has been found in many studies that extraverts show greater reminiscence and less vigilance than introverts (Eysenck, 1957; 1960b; 1962). The reader is referred to these references for a discussion of the large number of predictions so far tested within the framework of our theory.

Before turning to the postulated relationship between personality dimensions and drug effects, we must draw attention to one feature of our theory which requires special mention. By postulating a causal connection between inhibition and extraversion, we have for all practical purposes arrived at the postulation of two kinds of extraversion. Consider Fig. 2, which has been taken from Eysenck (1960d.) Here we have in diagrammatic form

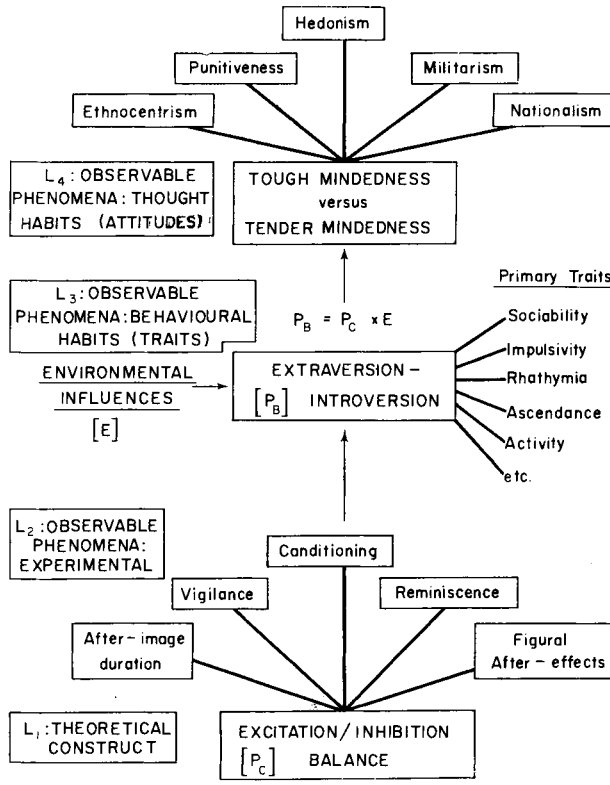


FIG. 2. Levels of Personality: Diagram illustrating a general Theory of Personality. From Eysenck, 1960.

a general picture of our theory. At the most fundamental level (L_1) we have the theoretical constructs of excitation and inhibition; these lead to certain observable experimental phenomena at the second level (L_2). These phenomena interact with environmental influences along the lines of our Fig. 1, and give rise to observable phenomena at level three (L_3), i.e. that of behavioural habits or traits. Finally these traits find specific expression in certain attitudes, which constitute yet another, higher level of observable phenomena (L_4). (The study of attitudes is not relevant to our purpose here, and will not be discussed further; cf. Eysenck (1954) for a full experimental account.) We may consider the L_1 construct as an essentially *constitutional* determinant of personality, and symbolize it as P_C; it must interact with *environmental influences* (E) to produce the L_3 type of phenomenon, which may be symbolized as P_B, or the *behavioural* traits of personality. The

fundamental equation linking these variables would be something like this:

$$P_B = f(P_C \times E);$$

in other words, the behavioural trait (or type) of extraversion – introversion is a function of the constitutional type and the environmental history of the organism.

We thus have two kinds of extraversion: E_B or behavioural extraversion, which is being measured by ratings and such devices as the Maudsley Personality Inventory (Eysenck, 1959), and E_C or constitutional extraversion, which is being measured to some degree of approximation by such tests as those listed in Fig. 2 under L_2. The degree of correspondence between E_B and E_C is very much dependent on E, the type of environmental influence brought to bear on the organism. Little empirical work has unfortunately been done in this field; nevertheless, on theoretical grounds this inclusion of E is vital for anyone concerned to make the correct predictions on to behavioural variables. One example may make this point clear.

Of particular importance in relating levels 2 and 3 is the theoretical conception of the *socialization process*, which is being mediated through some form of conditioning (Mowrer, 1950; Eysenck, 1957; 1959). According to this view, socialized behaviour in the adult has as its basis anxiety and fear responses to anti-social acts of an overtly aggressive or sexual character; these responses are conditioned in childhood and cohere together according to the principles of stimulus generalization (aided by verbal identification). It follows from the general theory of inhibition and excitation, as related to introversion – extraversion, that conditioning should proceed quicker, more strongly and more lastingly in introverts (Eysenck, 1957; Franks, 1956; 1957; Vogel, 1961), a prediction which has received much support. (cf. Fig. 3) It would also seem to follow that the barrier to the immediate satisfaction of every passing impulse (conscience, "inner light", super ego) which results from this socialization conditioning process should be stronger in introverts than in extraverts, due to the greater strength of the conditioning process in the former. Taken to extremes, this gives us the neurotic introvert, the dysthymic, who is over-socialized and prone to phobias and anxieties due to his over-strong conditioning equipment, and the neurotic extravert, the psychopath, who is undersocialized and prone to anti-social acts due to his defective conditioning equipment (Eysenck, 1960e). (This conditioned ethico-religious barrier to impulse satisfaction in the introvert also emerges in the attitude field in the form of "tender-minded" attitudes, while the relative absence of such barriers gives rise to "tough-minded" attitudes – Eysenck, 1954.)

If we apply these notions to criminality, and particularly to recidivist criminals, we might be tempted to argue that surely these should be overwhelmingly extraverted in personality, the reasoning being that extraverts, due to their defective conditioning equipment, had failed to become sufficiently socialized to avoid criminal behaviour. But such an argument would leave out of account the important differences in the actual conditioned and unconditioned stimuli brought to bear on different individuals

by their environment. Smith may be extraverted and difficult to condition, while Brown is introverted and easy to condition, but this does not

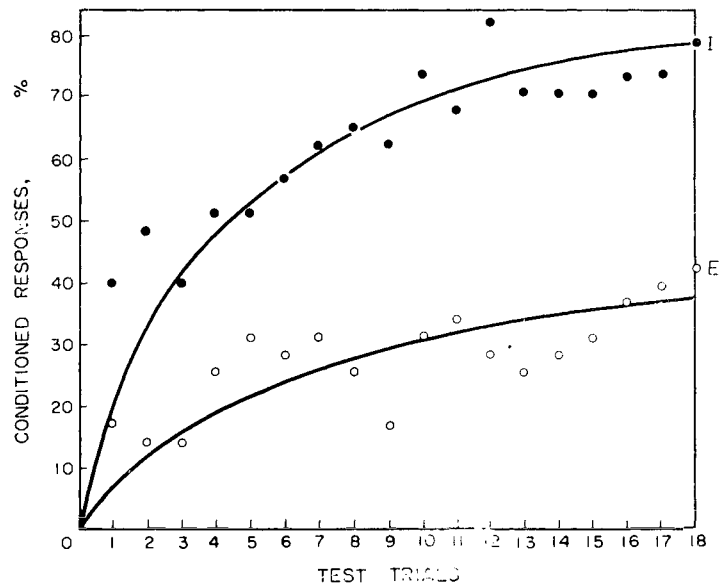


FIG. 3. Eye-blink conditioning in Introverts (I) and Extraverts(E). Eighteen test-trials are plotted; there are 35 normal and neurotic subjects in each group, the groups being matched for composition. The data are taken from two separate studies by Franks, cited in the text.

enable us to predict which of the two will acquire, say, a conditioned eyeblink response to a tone unless we know that both have been submitted to an identical conditioning procedure. If Smith has been put through 300 pairings of CS and UCS, and Jones has never been exposed to either stimulus, then clearly a different prediction would have to be made. Similarly, if Smith is brought up by parents who adhere strictly and undeviatingly to the rule "of spare the rod and spoil the child", and who in addition follow in all respects the rules and mores of the society in which they live, while Jones is brought up by a prostitute mother and a criminal father, both of whom neglect him and leave him to seek whatever social conditioning he may require from the neighbourhood gang of Borstal boys and spivs, then to predict on the basis of the subject's conditionability alone while leaving out of account completely the environmental determinants, would obviously be an act of crass stupidity. This should be too obvious to require mention, were it not that some critics have apparently misunderstood the

argument sufficiently to make such an explicit statement necessary. We may continue and point out that if most criminals come from a background which itself is criminal and therefore likely to condition the child born into it along lines which run counter to those of the larger society of which this particular Fagin's kitchen is only a small sub-group, then exactly the opposite prediction would follow: introverts should condition these criminal values more quickly and strongly and adhere to them longer and against all the efforts of the law and its agencies. Whatever may be the truth, clearly no simple answers are likely to present a solution to this age-old problem; as a minimum, P_C and E are both required to make use of our predictive equation. (Trasler, 1962)

To return now to our two kinds of extraversion – behavioural and constitutional. We would not expect anything in the nature of a perfect correlation between these two, for the simple reason that individuals of identical constitution may have quite divergent life experiences, i.e. have been exposed to entirely different conditioning situations, thus putting them at different places on the behavioural extraversion – introversion continuum. The best evidence we have been able to obtain comes from work such as that of Claridge (1960) who factor-analysed the intercorrelations between various experimental measures of P'_C, but also included the Maudsley Personality Inventory as a measure of P_B. Under such conditions the loading of the M.P.I. tends to be in the neighbourhood of ·6, which, if corrected for unreliability, seems to indicate that approximately half of the variance of P_B is accounted for by P_C, leaving approximately half to the influence of E. The argument is by no means rigorous, nor are the figures as reliable as one might wish, but in the absence of anything better, they may serve until more reliable and trustworthy figures are available.

These ideas lead to two main lines of research, neither of which has so far been at all widely adopted. In the first place, we would now appear to have available a method for investigating the effects of environmental influences on personality. The usual manner of demonstrating such dependence has been to study groups of children respectively showing and not showing certain behavioural characteristics; parental behaviour patterns on which the two groups could be differentiated were then supposed to be causally related to the child's personality development. Or conversely, groups of parents respectively showing and not showing certain behavioural characteristics have been studied; child behaviour patterns on which the two groups could be differentiated were then supposed to be determined causally by the parental behaviour pattern. Such demonstrations of *statistical correlations* clearly do not demonstrate *causal relationships* (Eysenck, 1960a) and alternative explanations are not only possible but likely (Eysenck, 1960f). One such explanation relates to the direct inheritance of constitutional predispositions; introverted parents may have introverted children, not because of environmental determination, but because of hereditary predisposition. The argument, which is universally conceded in relation to intelligence, is still widely neglected and disregarded in relation to other manifestations of personality. If now we are right in believing that an approximation to the measurement of P_C becomes possible in terms of the ex-

perimental tests at L_2 of Fig. 2, then it should also become possible to sort out the environmental and constitutional factors in experiments of the type described above.

However, in this book we are concerned rather with another line of research which also follows directly from our general theoretical framework. It is usually considered that a phenomenon has not been placed upon a secure scientific foundation until it has become amenable to experimental control, i.e. until it can be manipulated at will through changes in some independent variable which according to theory stands in direct relation to the dependent variable. The obvious difficulties of applying this paradigm to personality study have long served as an excuse for tolerating less rigorous methods, but it seems likely that advance will be much more rapid if and when this restriction can be removed. It is here that the drug postulate comes in according to which stimulant drugs increase excitation and therefore have an introverting effect, while depressant drugs increase inhibition and therefore have an extraverting effect (Eysenck, 1957). If this were true, then we would have here an experimental method of altering P_C (the dependent variable) by means of drug administration (the independent variable). This possibility seems of considerable importance to the writer, and he feels very strongly that a good deal of research endeavour could with great advantage be directed at this target. It may sound rather unusual to say that effects of certain drugs on laboratory measures of light sensitivity, conditioning, apparent movement, masking or vigilance may be of fundamental importance to personality theory, but is is of course common place in scientific research to find that the quickest way to a particular target may be the long way round, while the apparently direct way leads nowhere.

The general theory linking drug action and personality had been adumbrated by Pavlov (1927) and McDougall (1926), but they worked under the great disadvantage that little was known experimentally about the effects of inhibition; thus their exciting speculations did not result in any large body of experimentation. Hull (1935) also saw the importance of drug experiments in relation to the clarification of the concept of inhibition, but was discouraged by the failure of his main prediction to be verified. This prediction, namely that the bowing of the serial position learning curve should be increased by depressant drugs and decreased by stimulant drugs, also failed to be verified in our own work, and as many interesting points are raised by this failure, we shall come back to it later.

For the moment, consider the main types of research designs which are opened up by the drug postulate; they are shown in diagrammatic form in Fig. 4. Consider predictions such as: Depressant drugs will decrease conditionability, increase pain tolerance, increase alternation behaviour, decrease vigilance, elevate C.F.F. thresholds, shorten after-image duration and so forth. Clearly these follow the paradigm of Fig. 4 A, in that they do not depend in any way on the assessment of the personality of the subjects prior to the experiment. Subjects are randomly allocated to control and experimental groups and what is studied is the general effect of the drugs under investigation on what might be called the standard or average subject. The paradigm of Fig. 4 B is essentially different, in that it makes

use of the known position of groups of subjects (such as dysthymics and hysterics) on the introversion - extraversion continuum. This design harks back to McDougall's observation that extraverts need less alcohol to reach a point of intoxication than do introverts, who with the same amount of alcohol simply become more extraverted. Such a research design requires an objective *terminus ad quem*, i.e. the determination of an "intoxication threshold" which would enable us to ascertain the amount of alcohol required by different groups of subjects to reach the same level of cortical inhibition as defined by this threshold. Shagass's "sedation threshold" (discussed extensively in several chapters of this book) is an example of this.

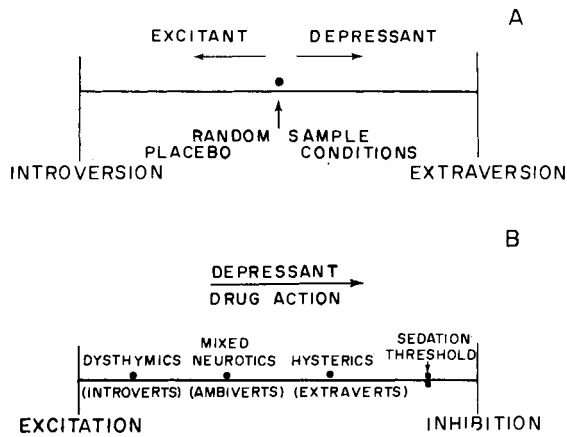


FIG. 4. Two paradigms of Drug Research, using stimulant and depressant drugs. From Eysenck, 1957.

Much work along both these paradigms has been done in our department and some twenty typical predictions have been listed in Table 1, together with references to books and papers describing experiments carried out specifically to test these predictions. In several cases work from other departments seemed so closely relevant that it has also been cited (Drew; Cohen *et al.*; Summerfield and Steinberg), and one or two classic studies from other sources could hardy be left out (Mackworth; Shagass). The list is neither complete as far as our own studies go nor does it pretend to do justice to the literature, which is of course quite large in connection with several of the topics mentioned (C.F.F.; conditioning); a survey of the literature has been attempted elsewhere by Trouton and Eysenck (1960). The Table is merely meant to illustrate the variety of experiments which are mediated by our theory, and the extensive support which such experimental investigations have given to the theory.

TABLE 1

Drug Studies

Topic	*Prediction:*		*References:*
	Stimulant	*Depressant*	
1. Conditioning	Increased	Decreased	Willet (1960) Franks & Laverty (1955) Franks & Trouton (1958)
2. Sedation threshold	Raised	Lowered	Shagass & Naiman (1956) cf. also chapter 5.
3. Vigilance	Increased	Decreased	Mackworth (1948) Felsinger, Lasagna & Beecher (1953) Treadwell (1960)
4. Rotation spiral after-effects	Increased	Decreased	Eysenck, Holland & Trouton (1955) Eysenck & Easterbrook (1960c)
5. Suppression of primary visual stimulus	Decreased	Increased	Eysenck & Aiba (1957)
6. Nonsense syllable learning	Improved	Worsened	Willett (1960) Eysenck (1957)
7. Motor responses	Smaller	Larger	Rachman (1961)
8. Time perception	Increased	Decreased	Costello (1961)
9. Kinaesthetic figural after-effects	Decreased	Increased	Poser (1958) Eysenck & Easterbrook (1960)
10. Pupillary responsiveness	Increased	Retarded	Eysenck & Easterbrook (1960e)
11. Static Ataxia	Decreased	Increased	Holland (1960) Eysenck & Easterbrook (1960a)
12. Adaptation (palmar skin resistance)	Weakened	Strengthened	Martin (1960)
13. Critical Flicker Fusion threshold	Depressed	Elevanted	Holland (1960)
14. Alternation behaviour	Decreased	Increased	Sinha, Franks & Broadhurst (1958)
15. After-image duration	Lenghtened	Shortened	Chapter 7
16. Visual field sensitivity	Heightened	Reduced	Holland (1960)
17. Pursuit rotor performance	Increased	Decreased	Eysenck, Casey & Trouton (1957)

Topic	*Prediction:*		*References:*
	Stimulant	*Depressans*	
18. Apparent movement threshold	Increased	Lowered	Chapters 10 and 11
19. Visual masking effects	Decreased	Increased	Chapters 2 and 3
20. Set	Increased	Decreased	Martin (1960)
21. Risk taking	Decreased	Increased	Cohen et al. (1958)
22. Performance accuracy	Increased	Decreased	Drew at al. (1959)
23. Retroactive interference	Increased	Decreased	Summerfield & Steinberg (1957)
24. C.E.R.	Increased	Decreased	Singh (1959; 1961) Singh & Eysenck (1960)

In looking at the various different topics investigated, it should be realized that the chain of deductions from theory to experiment may greatly vary in length or solidity. It is often assumed that a theory "generates" a deduction, but such a statement is only partially true (Eysenck, 1960b). Given that certain other facts and theories are true, fairly firm deductions may indeed follow from a given theory, but if these assumptions are not in fact correct or if the hypotheses and facts external to the theory under investigation admit of different interpretations, then the whole method becomes very much more complex. Consider as an example the prediction by Hull (1935), already mentioned, according to which depressant drugs should increase the bowing of the serial position learning curve, while stimulant drugs should decrease it. This prediction, as well as a similar one by Eysenck (1957) that extraverts should show greater bowing than introverts, was based on the assumption that the Hull–Lepley theory regarding the origin of this bowing effect was correct, and that certain inhibitory potentials were produced during learning and accumulated more in the central positions of the list than at the ends. The failure of Hull (1935) and Willett (cf. Eysenck, 1957) to find the predicted differential bowing effects after drug administration and the failure of Carpenter (1960) to find it in relation to personality differences, suggests the possibility that the Hull–Lepley hypothesis might in fact be wrong; in a specially designed experiment Eysenck (1959) was able to show that certain empirical results were indeed incompatible with this hypothesis. It follows that Hull's original prediction, as well as those later made by Eysenck did not in fact represent correct deductions from the drug and personality theories under investigation because of the failure of the link between these theories and the theory underlying the phenomenon in question.

Consider next the phenomenon known as "reversible perspective alternation", which is discussed in some detail in this book by Sylvester. McDougall (1924) suggested originally that reversals should be more numerous in introverts than in extraverts, his argument apparently resting on what we would now call the facilitating effects of greater excitatory potential of

introverts. More recent writers have suggested that alternation effects of this type are due to perceptual satiation, which has theoretical affinities with inhibition; it would follow that greater satiation, leading to a more rapid rate of alternation, would be found in extraverts, and generally as a consequence of depressant drugs.* We thus have two contradictory predictions from our drug and personality postulates, depending on the theory which we choose to hold regarding the experimental phenomenon in question. No crucial experiment is possible under the circumstances, as an important link in the chain of reasoning is weak and unreliable. The fact that Eysenck, Holland and Trouton (1957) found no drug effects either way may suggest that both alternative theories are in fact wrong or it might even be that extraverts and introverts show effects going in different directions. Clearly greater clarification of the phenomenon itself is required before the results can be used either to support or weaken our theory.

Readers of the various experimental chapters in this book will find this point streessed again and again. A given experiment is carried out because according to some theories at least the phenomena occuring in that experiment are related to the concepts of excitation and inhibition. Usually the proof for such a hypothesis is indirect, often doubtful, and seldom of a kind which would definitively settle the issue. Under the circumstances, therefore, predictions cannot be made with any very great confidence and their verification depends on the truth of the hypothesis held with respect to the phenomena under investigation. It therefore becomes important to investigate these phenomena themselves and much of our work has been devoted to the direct experimental study of such experimental facts as the suppression of the primary visual stimulus, in the hope that we would be able to put our predictions on a more satisfactory footing. Indeed the writer is convinced that the traffic between experimental and theoretical psychology on the one hand and drug and personality study on the other, is by no means one way. It would be quite untrue to say that experimental and theoretical psychology furnish an established framework which unambiguously mediates predictions made on the basis of our personality and drug postulates. The failure of such predictions as those of Hull and Eysenck with respect to the bowing of the serial position learning curve must react adversely on the theory of that effect and lead to reconsideration and new experiments such as those of Eysenck (1959). Similarly the failure of depressant drugs to increase the rate of alternation of reversible perspective figures must throw doubt on the explanation of this effect in terms of satiation. Many other examples of such interactions are given in the review of psychopharmacological studies by Trouton and Eysenck (1960), and the writer finds

* Some recent work by Fisichelli (1947) and Lynn (1961) indicates one way in which cortical inhibition might be used to give predictions in the same direction as McDougall's. These writers have shown "that rate of reversal is a function of the amount of stimulation by showing that reversal rate is a function of stimulus-intensity". Now the inhibition hypothesis and Broadbent's "filter" hypothesis predict a *lowering* of stimulus intensity over time, and this would be more pronounced in extraverts than in introverts. It is of course possible that both applications of the inhibition hypothesis are correct, in which case they would be cancelling each other out. Clearly this is a particularly complex phenomenon requiring much further research for its elucidation.

it surprising that more use has not been made by experimental psychologists of the important property of drugs temporarily to alter the properties of the organism which mediates between stimulus and response. Possibly this neglect is due to the widespread failure of S-R psychologists to recognize the absolutely fundamental importance of the organism which should, properly speaking, intervene between them to make behaviour $=(f)$S-O-R.

A special word should perhaps be said here about the reminiscence effect, which is not mentioned among the topics in Table 1. Several experiments (Eysenck, 1960b) have failed to demonstrate drug effects on reminiscence and this may be thought to invalidate our general theory in view of the fact that reminiscence is undoubtedly an inhibition-produced phenomenon. However, the facts are a little more complicated than this. The theory (Eysenck, 1962) states that inhibition will be produced more rapidly and dissipate more slowly in extraverts, and after the administration of depressant drugs; this means that differential effects are difficult to observe unless the timing of the experiment is adjusted so precisely that: (a) the rest pause is introduced at a point when inhibition is still growing and has not reached its asymptote yet; and/or (b) the length of the rest pause is such that complete dissipation of inhibition is not achieved. It is very doubtful if existing experiments have come anywhere near fulfilling these conditions and consequently we can at the moment come to no definitive conclusion about this question. Again it must be stated that here also research is very much handicapped by insufficient knowledge of the parameters underlying manifestations of the phenomenon in question. Much further work will have to be done before these issues can be clarified.

There would be little point in discussing in turn such apparent failures of prediction as in the case of auditory flutter fusion (Eysenck and Easterbrook, 1960f), or visual figural after-effects (Eysenck and Easterbrook, 1960d), or to comment in detail on revisions of the theory made necessary by the discovery of additional facts, as in the case of flicker fusion thresholds (Eysenck and Easterbrook, 1960f). These have all been discussed in their proper place and suggestions made regarding the possible reasons for the failure. For reasons the writer has discussed elsewhere (Eysenck, 1960b), it is much more interesting and important, in the case of weak theories (such as theories in psychology are apt to be), to concentrate on successful rather than unsuccessful predictions. The reasons for failure are numerous and may be difficult to disentangle. They certainly do not necessarily implicate the specific theory which is being tested. The reasons for success may also of course be numerous and may not necessarily imply support for the theory which gives rise to the prediction (Broadbent, 1961). However, when a long list of successful predictions can be drawn up (as has been done in the case of Table 1), it becomes rather more difficult to explain them all away in terms of accidental factors or factors extraneous to the theory. It is, of course, possible, though not perhaps likely, that each single experiment can be explained in terms specific to itself and without implications for the other experiments listed. It seems more likely that some general feature does indeed link all these different phenomena, and is being influenced in a systematic fashion by all the drugs used. Our theory may not be

correct as it stands, but it is likely to contain certain features which will have to form part and parcel of any theory which may supersede it.

We must now turn to a criticism which will have occurred to readers familiar with the pharmacological literature. We have referred quite blandly to "depressant" and "stimulant" drugs, without any further discussion of the pharmacological properties associated with these two groups or the principles according to which a drug would be classed in one group or the other. Nor have we discussed the important question of side effects, peripheral action etc., yet it is well kown that there is no general agreement on these problems, and that the terms "depressant" and "stimulant", while having wide circulation, do not enjoy any precise definition (cf. chapter by D. Gooch). What we appear to have done, it might appear to the reader, has been to take loosely defined concepts from pharmacology and relate them to loosely defined concepts in experimental psychology through the *pons asinorum* of even more loosely defined concepts in personality theory! Such a criticism would not be entirely just, but it does incorporate enough truth to require discussion. The writer would put the matter like this. Psychology and the sciences associated with it have reached only a very elementary level of explanatory power and of predictive accuracy. None of the theoretical concepts currently fashionable are likely to last, and many of them are liable to be superseded within a matter of years. This is a position which no amount of argumentation will alter or ameliorate. The only way out is to recognize quite consciously the unsatisfactory nature of the concepts and the nature of the evidence supporting them and to realize that *rigour* in the sense in which the word is understood in mathematics and physics is simply not possible in psychology at the present time. This does not mean that we should give up efforts to be as scientific and rigorous as possible; it means simply that we should recognize that standards of perfection which are appropriate to a mature science may not be possible of accomplishment in a young and immature one. *Impossibilium nulla obligatio.*

This situation seems to require a certain type of experimental procedure, which would lead to the mutual strengthening and interaction of similar concepts usually studied in isolation. By drawing them together in a common "nomological network", one set of facts and theories may be able to throw light on the others and in return receive further clarification itself. Search for further experimental data is stimulated and concepts, theories and experiments should acquire an ever growing rigour and precision. The writer finds it difficult to see how personality research can successfully achieve its object whilst continuing to disregard almost completely the contribution of experimental psychology; similarly he feels strongly that by rejecting the facts of systematic individual differences, experimental psychology is deliberately sacrificing a vitally important part of the data on which the science of psychology must be built. It would perhaps not be too far-fetched to say that one of the reasons for the relative poverty o psychology lies in its premature compartmentalization. It is one of the aims of this book to try and break down this parochial approach and to suggest ways in which fruitful interaction is possible.

Let us consider just one way in which this could be done. We start out with a vague and clearly unsatisfactory concept, such as that of "depressant" drugs. Apart from the fact that this group is not clearly defined, we do not know how it is related to hypnotics, to sedatives, to tranquillizers, and to many other hypothetical groups, which may or may not be wholly or partly identical with "depressants". We now proceed to formulate a hypothesis regarding the action of these drugs, stressing the increase of inhibitory potential and the decrease of excitatory potential. These concepts are anchored more or less securely in a large number of experimental phenomena such as those listed in Table 1. We can now go through a few representative "depressant" drugs to see whether in fact they have the predicted effects. Finding that predictions are verified in the great majority of cases we can now proceed to *define* the term "depressant" drug operationally in terms of specific effects in clearly specified experimental situations; this can be done with considerable rigour by the use of modern mathematical methods (Eysenck and Eysenck, 1960). We can conversely define the concept of "inhibition", as used to explain certain features of the experimental situation, by relating it to the effect of "depressant" drugs. The net can be widened again by linking both experimental and drug effects with the personality dimension, extraversion – introversion, along similar lines, and by linking all three with brain damage, old age etc. In this way, and the writer would almost be inclined to say, only in this way, is it likely that each constituent part of the general scheme can transcend its natural limitations. In this argument we have been concerned only with one particular dimension of personality, but the argument is, of course, perfectly general and applies to all dimensions of personality (Trouton and Eysenck, 1960; Eysenck, 1960i). Futhermore, the argument goes beyond the confines of psychology and may lead to closer integration with physiological principles, as for instance in the hypothesis that "inhibition" as used in psychology may be linked with "inhibition" as conceived by physiologists working on the ascending reticular formation. This whole process of give and take is, of course, an extremely complex one, but it does seem to the writer to embody the essence of the hypothetico-deductive method which has been so enormously fruitful in the older sciences (Hanson, 1958). It is easy to say, in relation to any particular deduction or experiment, that it is not conclusive, or that it is not sufficiently rigorous, or that much further work will be required; such criticisms are not really useful because they inevitably apply to all psychological research, and indeed to all scientific research. It may be recalled in connexion with one of the strongest theories ever enunciated that Newton accounted for certain errors in his predictions by postulating that angels were pushing the planets off their courses, and that the whole mathematical technique of the infinitesimal calculus only became universally accepted and rigorous 150 years after Newton, when Cauchy wrote his *Cours d'Analyse*. Even in physics and mathematics, progress, particularly in the early stages, has been agonizingly slow and full of wrong turnings and cul-de-sacs. Psychology undoubtedly will not be free of similar set-backs, but it is likely to overcome these provided that it does not set its sights too high at too early a time in its development.

Chapter 2

THE SUPPRESSION OF THE PRIMARY VISUAL STIMULUS

S. AIBA*

BACKGROUND TO THE PROBLEM

(1) *Introduction*

This is a report of an investigation of visual after-images as seen in the form of the Bidwell phenomenon.

This phenomenon, which was first demonstrated by Bidwell (1896, 1897) may be described as the suppression of the primary visual stimulus *and* simultaneous production of the negative after-image of the stimulus suppressed.

Suppression of the primary stimulus can occur without the production of negative after-images as in the case of meta-contrast (Stigler, 1910; Baumgardt and Segal, 1947; Alpern, 1953; and many others); likewise, the negative after-image can occur without suppression of the primary stimulus as in the usual situation in which ordinary negative after-images are observed. Neither of these is studied in the present investigation.

It is difficult to generalize the conditions under which the phenomenon in question can be observed, but on the basis of the experimental works done since Bidwell's time, they may be safely summarized as follows: The stimuli involved in the occurrence of the Bidwell phenomenon are two short flashes applied to the eye, one immediately after the other, i.e. without a dark interval between them. The first flash is normally in colour and is given for a relatively short period (5–50 msec); the second one is invariably white and somewhat longer in duration (50–100 msec). These flashes are preceded by a dark period lasting for no less than 10 msec. In most instances, the first of the two stimuli (i.e. the coloured one) is presented with a white surrounding field, and the second one (i.e. white) generally covers an area about the same size as this surrounding field. Thus, the second stimulus (white) covers an area which is *always* larger than the coloured central part of the first stimulus. The coloured stimulus is usually centrally fixated. The retinal area stimulated by the coloured part of the first stimulus is generally confined within a radius of about 1° from the centre of the fovea, but the area stimulated by the second stimulus, and by the white background of the first stimulus, may exceed a 2° or 3° radius

* The writer is indebted to the Wallace Laboratories for the support of this investigation. He is now at the Psycho-Acoustic Laboratory, Harvard University.

from the same spot. As a rule, luminances of both stimuli are at photopic levels and observations are made with an eye which is light-adapted to various extents.

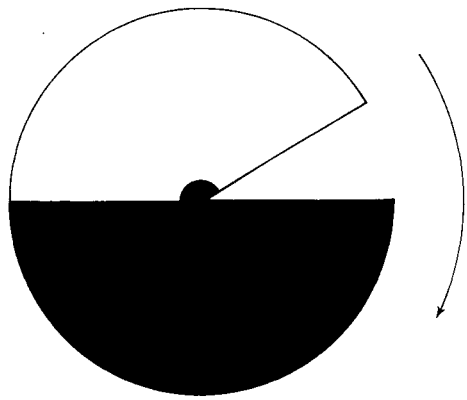


FIG. 1. Bidwell's disk. The arrow shows the direction of rotation to obtain the effect.

In Bidwell's original demonstrations, the above stimulus conditions were produced by means of a rotating sector disk. The disk was divided into 2 equal parts of black and white by a straight line passing through the centre of the disk and a sector (15–30°) cut in it along the dividing line (see Fig. 1). A stimulus painted in colour on a white paper was seen through the disk which was rotated at, say, 5 r.p.s. while both the paper (on which the stimulus is painted) and the disk were sufficiently illuminated. When the direction of the rotation was such that a part of the retina of the observer's eye was first stimulated by the black area of the disk, then by the coloured stimulus (through the sector) and finally by the white area of the disk, suppression of the coloured stimulus occurred and a sensation of a complementary colour was experienced instead.

There may be many ways in which the Bidwell phenomenon can be studied. The present author, however, was throughout concerned with the determination of the ranges of intensity* of the two stimuli required to produce the phenomenon. As will be seen, the production of the phenomenon depends upon *relative* intensities of the two stimuli as well as their *absolute* levels.

All the measurements in this study were based on the judgement of whether or not the Bidwell phenomenon was observed, and the criterion for the seeing and not-seeing of the phenomenon (i.e. the presence and absence of it) was simply based on whether the suppression of the primary stimulus was complete or not. Thus when the hue of the primary stimulus was seen (however faintly), the suppression of the stimulus was considered

* By "intensity" is here meant the product of luminance and duration of the stimuli. In brief flashes, the two variables are reciprocal. (Bunsen–Roscoe law).

as not complete, and, therefore, the perfect Bidwell phenomenon was considered as absent; and vice versa.

(2) *Recent studies*

Although visual after-images as such have received considerable attention, the particular type of after-image phenomenon which we are investigating has been relatively neglected. There have been, however, a few experimental studies on the phenomenon in the last decade or so. They have all used basically the same technique, i.e. Lehmann's (1951) device for measuring the threshold of the phenomenon. Lehmann constructed an apparatus similar to the sector disk arrangement which Bidwell had originally used, but, in addition to it, provided a pair of Polaroid filters between the disk and the observer's eye. The function of these filters was to reduce the luminances of the disk and the stimulus (which was painted on a white paper and placed behind the disk) by any desired amount. Lehmann showed that, when other conditions were kept constant, reduction of the luminances of the disk and the stimulus by the Polaroid filters immediately destroyed the phenomenon which had been seen clearly before. Thus he could obtain a kind of threshold for the appearance and disappearance of the phenomenon (i.e. the threshold for negative after-images) by denoting the density of the filters at which the phenomenon disappeared. He called this the after-image disappearance threshold.

After standardizing the measures obtained by this method in regard to both age and sex (Csank, 1955), Lehmann and Csank (1957) used the same technique to study the effects of various drugs on the subject's "basic mental processes" in conjunction with 8 other psychological and psychophysiological tests. He found that the after-image disappearance level was particularly sensitive to the action of drugs. Critical flicker fusion frequency was also found to be quite sensitive, and, in most cases, appeared to be affected by drugs in a parallel way with the after-image threshold.

Levinson (1952) made use of Lehmann's technique to study relations between certain aspects of personality and perception. He failed to find any relations between the negative after-image threshold as measured by this method and any measures of personality, or any other perceptual measures which included critical flicker fusion frequency and negative after-image duration.

A slightly different use was made of Lehmann's method in a series of experiments conducted by Kaplan (1960, a,b). Prompted by Kravkov's (1941) earlier findings on the effects of sympathetic activities of the subject on his colour vision thresholds, he tried to find out whether the negative after-image threshold would suffer the same type of modification as the colour vision threshold under sympathomimetic and para-sympathomimetic drugs. He therefore tested the effects of ephedrine and prostigmine on the negative after-image threshold, using various colours. He found that both drugs changed the negative after-image threshold in a similar way, but that the magnitude of the effects was different for different colours, it being greatest for *red* under ephedrine, but greatest for *green* under prostigmine.

As this was more or less what could be expected from Kravkov's original findings, he concluded that Lehmann's method is an efficient tool for assessing sympathetic activities. In a subsequent study, he showed that people with high hypothalamic activity (as tested by Funkenstein mecholyl test) exhibited less of the above mentioned differential threshold effects under prostigmine than those with low hypothalamic activity. He attributed this to higher sympathetic activity in the former group of subjects, which counteracted the effect of prostigmine.

Original though the ideas behind these studies may be, one cannot help noticing rather inadequate control of the experimental conditions in some of the above cited studies. In none are the visual angles subtended by the stimuli or the position of the subject's fixation or the state of adaptation of the eye explicitly specified. In all but those of Kaplan, the luminances of the stimuli are given only in relative values, thus making assessment of the physical conditions virtually impossible. Control of subject's pupil size, by means of an artificial pupil, is also neglected, again with the exception of Kaplan's study. Thus, we are informed of very little about the experimental conditions under which the above studies were carried out, and have to depend largely on guesses to make any kind of generalization. Under these circumstances, it is impossible to assess the true significance of the above quoted studies, since the reported effects might be due to irrelevant factors.

THE APPARATUS

(1) *The apparatus for studying the Bidwell phenomenon*

Essentially, the apparatus used in the present study consisted of two light sources, one providing illumination for the first (colour) stimulus and the other for the succeeding (white) stimulus (this latter also provides illumination for the white background of the first stimulus), and a rotating disk to control the durations of the two stimuli. A schematic diagram of the apparatus is shown in Fig. 2a.

As can be seen from Fig. 2, a projector lamp, S 1, illuminated a thin frosted glass piece, G, the diffusely radiating surface of which constituted the first light source. A lens, L, formed the image of the source on the opposite side at G′, where an artificial pupil, AP, 3 mm in diameter was placed. The disk, placed at E, had a small aperture cut in it and, when rotated by a synchronous motor, M, at a fixed speed of 15 r.p.m., caused the light from G′ to reach and focus at G for a very brief period once every 4 seconds. A colour filter (Ilford 609 Spectrum Deep Red) was inserted at F in the path of the light. Thus, the subject whose eye was placed at AP would see brief red flashes repeatedly at fixed intervals. On the disk and immediately adjacent to the aperture was a white sector (see Fig. 2b), which was illuminated by the sources, S 2 and S 3. The function of this sector was both to cut off the light from G and at the same time to provide the white stimulus when the disk was rotated in the direction shown by the arrow in Fig. 2b. A tube,T, was placed between E and AP, which, in conjunction with a screen,

SC 1, prevented the light from S 2 and S 3 from reaching the subject's eye directly. The tube, T, also served to restrict the size of the white stimulus. The size of the red stimulus was limited by a circular diaphragm, D, which was placed immediately behind E. Both the light from G and the reflected light from the white sector on E could be occluded by a screen, SC 2, when this was lowered just in front of the disk E. On the surface of the screen facing toward the subject, a paper having various reflectances could be attached so as to provide an adaptation field having various luminances when it was lowered and its surface illuminated by the sources, S 2 and S 3. The fixation point was provided by a crossed hair reticle at FP which indicated the common centre of the two circular stimuli.

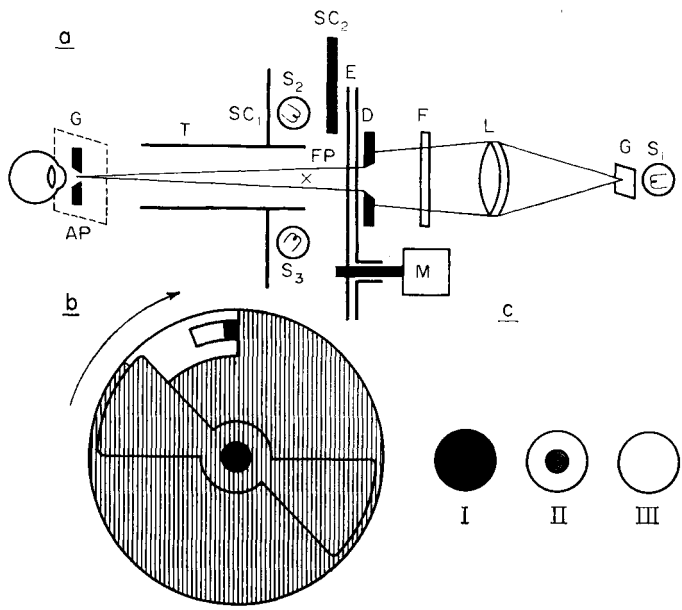


FIG. 2. The schematic diagram of the apparatus for studying the Bidwell phenomenon. For explanation, see the text.

The duration of the first stimulus was varied by enlarging or reducing the size of the aperture in the disk. This was achieved by moving a metal piece attached to the disk so as to block various proportions of the existing larger aperture. The duration of the second stimulus was varied by another metal piece painted black which could be moved around to cover various degrees of the white sector (see Fig. 2b). The sizes of both stimuli were kept constant throughout the present investigation.

Thus, the first (red) stimulus always subtended a visual angle of 1°, and the second (white) stimulus a visual angle of 4°. It should be noted, however, that the first stimulus was presented all the time with a white annular surrounding field. This annular field was provided by the diaphragm,

D, which was, it will be remembered, primarily placed there to limit the size of the coloured stimulus; the diaphragm was made of white paper and was illuminated, through the aperture in the disk, E, by S 2 and S 3, at the time when the coloured stimulus was presented, thus providing the white background to this stimulus. The outer circumference of the annular surrounding field thus produced coincided with that of the second stimulus (hence, being 4° in visual angle) by virtue of the tube, T, through which both stimuli were observed.

The stimuli were presented in the order I, II and III of Fig. 2c in which the period of dark is represented by I, the red (coloured) and the white stimuli by II and III, respectively.

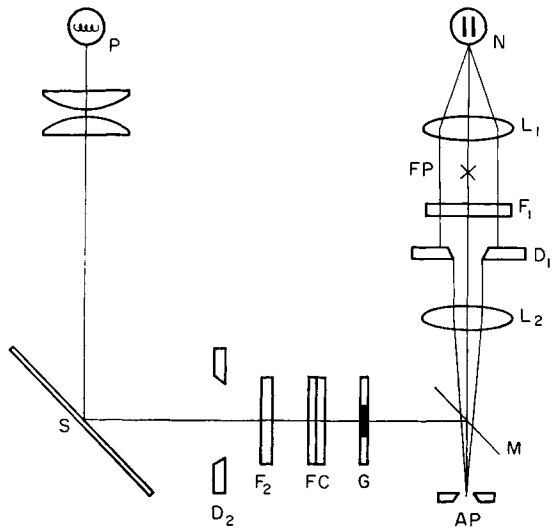


FIG. 3. The schematic diagram of the optical system used in the flicker apparatus. For explanation, see the text.

The luminance of the red stimulus was varied by means of a variac which controlled the voltage supplied to the projector lamp. Changes in the spectral quality of the stimulus produced by the changes in the voltage supplied to the source were negligible since the light from the source was, before reaching the eye, transmitted through the monochromatic red filter which absorbed most of the wave-lengths of light in the human visual spectrum except a narrow band at the red end. Relative values were used in specifying the luminance of this stimulus, but it may be stated that 2·1 (log) in our units of luminance roughly corresponded to 2000 mL. The luminance of the second (white) stimulus was kept constant except in one study where it was varied over a range of 0·3 log unit. In the latter case, either the current supplied to the sources, S 2 and S 3, was altered, or neutral filters of various densities were inserted between AP and E (in Fig. 2a.)

When the latter method was used, the luminance of the red stimulus was also reduced; it was, therefore, necessary to correct the readings for the luminance of this stimulus afterwards. The luminance of the white annular surround for the red stimulus was estimated to be the same as that of the second (white) stimulus, since the reflectances of both materials were identical, and E and D (in Fig. 2a.) on which they were affixed were situated nearly at the same distance from the sources, S 2 and S 3.Finally, the darkness of the dark period (Fig. 2c, I) is specified as a luminance of about 1/100 of the luminance of the white stimulus. Therefore, it varied with the luminance of the white stimulus. The variation, however, was very small compared with that of the luminance of the white stimulus in absolute values.

(2) *The apparatus for measuring critical frequency of flicker*

A schematic diagram of the apparatus is shown in Fig. 3, from which it will be seen that it consisted of two parts, one system for providing the illuminated background and a second system for providing the flickering source. The subject, with his eye placed immediately behind the artificial pupil, AP, viewed the flickering source, N, directly and the background (surround) by reflection in a very thin glass plate, M, placed at 45° to his line of regard. The part of the background corresponding to the flickering field was occupied by a black circle painted on a plain glass plate placed at G, so that the luminance of the flickering field could be varied independently of the surround illumination. The illumination for the surround was provided by a 250 watt projector lamp P, which, in conjunction with the projection lenses, uniformly illuminated the surface of a screen, S, 4 in. x 6 in. in size, which was made from a thin aluminium plate and was covered evenly with deposited magnesium oxide coating. Luminance of the surrounding field was dependent upon the angle of incidence of light from the projector lamp upon the screen (Lambert's law). In the present experiment this angle was fixed at 40°.

As the coating of magnesium oxide gives a nearly perfect diffuse surface, the direction from which the screen is viewed does not matter very much, although, in the present apparatus, the angle between that direction (after being deflected at M) and the direction of the light from the projector lamp was kept at 90°. In addition to this, a neutral density filter was inserted at F 2 when a *dim* surround was required during the experiment. The size of the surround was limited by a diaphragm, D 2 and its colour temperature was raised by a combination of Wratten Light Balancing Filters (82 B, 82 C) at FC. The illumination for the flickering source was provided by a Mazda Type-C neon lamp, N. Light from N was collimated by the lens, L 1; the parallel beam was then picked up by a second lens, L 2, which converged it to a focus in the plane of the artificial pupil, AP. An eye placed behind AP thus saw the lens, L 2, uniformly filled with light. The size of the flickering source was limited by a diaphragm at D 1, while its luminance could be varied by a neutral density filter at F 1. A fixation point was provided by a small black spot painted on a clean glass plate inserted at FP. The flicker

was generated by an electronic device (see Fig. 4; the circuit consists of a blocking oscillator providing pulses applied to an Eccles-Jordan bi-stable multivibrator) which was connected to the neon lamp. The light–dark ratio thus produced was approximately 1 to 1 over the entire range of frequency.

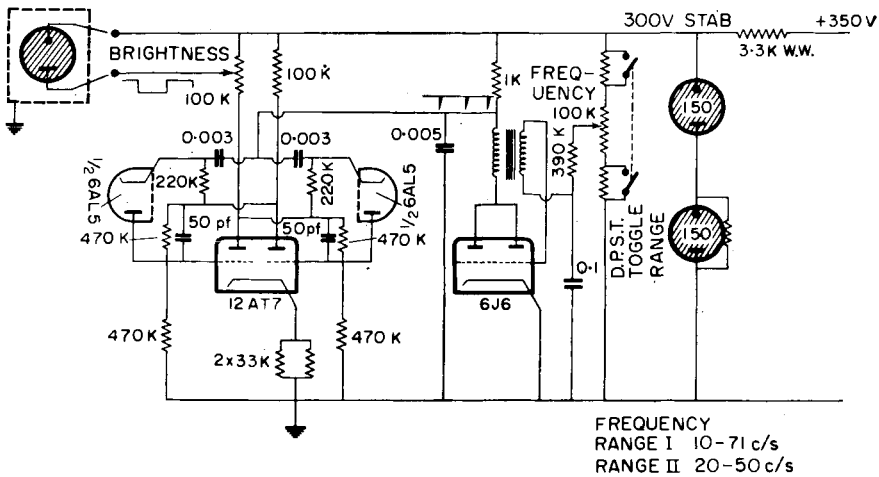


FIG. 4. The circuit diagram of the electronic flicker generator.

FUNCTIONAL RELATIONSHIPS BETWEEN THE VARIABLES

(1) *Introduction*

The conditions under which the Bidwell phenomenon can be observed have been delineated in the introductory section. As pointed out, there has been as yet no systematic attempt to specify these conditions in any quantitative manner. When the phenomenon was studied, first the stimulus values were usually sought in an arbitrary manner in order to produce the phenomenon, and then one or two of the variables involved were varied without reference to the physical characteristics of the original stimulus (or stimuli). By this method, one could get some kind of measures but would never know quite what they meant. Furthermore, when one followed this method, one would never be sure whether the results obtained were arbitrarily dependent on particular conditions which happened to be employed in the experiment, as was the case with some of the drug studies mentioned earlier. All this indicates that we must not only know the physical characteristics of the stimuli but also have a knowledge of the parameters involved in the production of the Bidwell phenomenon, if we are to carry out any meaningful experiment. For this purpose, the stimulus values will have to be specified in the accepted photometric and other

physical units and the stimuli concerned will have to be changed over wide ranges and their inter-relationships be determined, using such a criterion as has been described in the introductory section. The psycho-physical experiments to be reported in the following sections were carried out to satisfy these requirements.

(2) *Method*

Owing to the time required to make all the necessary measurements involved in the experiment, it was decided to split them into several parts, each part being carried out on a separate day.

The standard procedure for any such day was as follows: Following 15 min preliminary dark adaptation, the subject, with one eye covered and the other eye placed immediately behind the artificial pupil, saw the light adapting field having a luminance of 4·85 mL for two min. The adapting field was then removed and the subject was given the first stimulus sequence, and was asked what he had seen. The stimulus sequence, like any other one to be presented in the course of the experiment, consisted of a period of darkness lasting about 2·5 sec, the red flash lasting 4 msec and the white stimulus, the duration and the luminance of which were varied according to the condition being investigated. In the first sequence, the luminance of the red stimulus was such that the subject invariably saw only *green*. As soon as the first sequence had been presented, the adaptation field was lowered again and the subject looked at this for 5·3 sec. Then it was removed, and he was given the second stimulus sequence in which the luminance of the red stimulus was slightly higher than that of the first one, and he was asked to report what he had seen. He was given the third, fourth and all subsequent sequences in the same way, and the luminance of the red stimulus was progressively increased in each sequence at a fixed interval until the "threshold" was finally reached. The threshold was defined as 3 successive reports of any trace of *red* in the sequences presented. Prior to this, the subject saw various degrees of green. The measure taken was the luminance of the red stimulus which produced the threshold responses and it was recorded after each assessment (the recording was made automatically by means of a recording milli-ammeter which was connected to the projector lamp, as well as by the experimenter). The measurement was repeated three times with the same white stimulus, and the mean of these three estimates represented the measure (for that day) of the white stimulus under investigation. After a threshold had been determined the duration and/or the luminance of the white stimulus was changed, and the same procedure was repeated again, except for the initial dark adaptation period which was now replaced by a 2 min rest in the dark.

There were altogether 44 different conditions investigated, i.e. those characterized by 11 durations, combined with 4 luminances of the white stimulus. The 11 durations were 1·5, 1·55, 1·6, 1·65, 1·7, 1·75, 1·8, 1·85, 1·9, 2·0 and 2·1 log-msec., while the four luminances combined with these durations were 1·8, 1·9, 2·0 and 2·1 log-mL. The above described "threshold" measurements were made for each one of the 44 conditions. Any one of the 4

luminances in conjunction with the 11 durations was generally studied on a single day. Another session for testing exactly the same conditions (a replication) took place some days later.

(3) *Subjects*

Altogether 4 subjects were used in the present investigation. The main body of the study was, however, carried out with one highly trained subject (S.A.). Results of other subjects are presented for comparison and also for establishing the generality of S.A's results. The subject S.A. was a research psychologist, but the other 3 were all in non-psychological occupations and had no previous experience of being subjects in psychological experiments. None had gross anomalies in vision and all had a visual acuity of 6/9 or better as tested by a Snellen chart. In the case of S.A., who was myopic, the refractive error was corrected by a microcorneal contact lens.

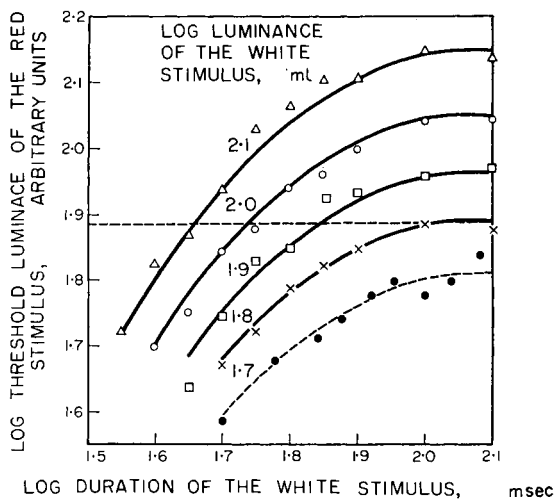


FIG. 5. Curves describing the relationship between the duration of the white stimulus and the threshold luminance of the red stimulus for various luminances of the former. A curve was first fitted to the points representing the data for the white stimulus of 2·0 log-mL the second fro m the top) by eye, and the same curve was then fitted to all other points. Subject: S.A.

(4) *Results*

The results of S.A. are given in Table 1, and those of the other subjects in Table 2. In Figs. 5 and 6 the same data are presented graphically. In these graphs, only averages are plotted for the sake of clarity. (Note, however, that each figure in the tables is based on at least 5 estimates.) The blank blocks in these tables indicate that suppression of the red stimulus did not

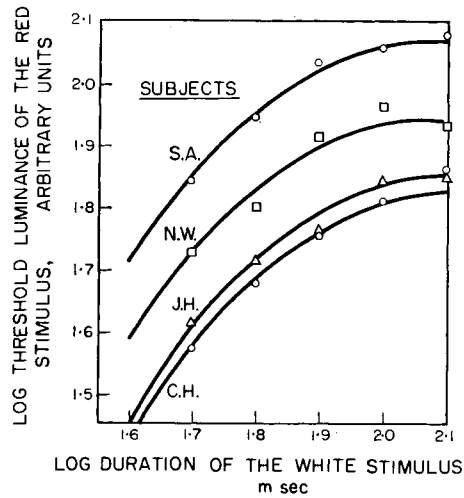


FIG. 6. Curves describing the relationships between the duration of the white stimulus and the threshold luminance of the red stimulus in 4 different subjects. The curve which fitted the data-points of S.A. was used to fit those of the other subjects. The curve for S.A. is moved upwards by 0·5 on the ordinate scale, to avoid confusion. True ordinate values of this curve may thus be read by subtracting 0·5 from the ordinates of the curve.

TABLE 1

Log threshold luminance of the red stimulus obtained with the white stimulus at various luminances and duration. Each value is based on 6 estimates

Duration of the white stimulus in log-msec.	*Luminance of the white stimulus in log-mL.*			
	2·1	2·0	1·9	1·8
1·5	–	–	–	–
1·55	1·720	–	–	–
1·6	1·823	1·698	–	–
1·65	1·867	1·750	1·631	–
1·7	1·936	1·845	1·743	1·607
1·75	2·027	1·873	1·832	1·719
1·8	2·061	1·937	1·846	1·787
1·85	2·101	1·953	1·931	1·287
1·9	2·100	1·997	1·930	1·843
2·0	2·143	2·034	1·952	1·882
2·1	1·130	2·040	1·971	1·873

TABLE 2

Log threshold luminance of the red stimulus obtained from the three subjects. The white stimulus: luminance 1·9 log-mL, duration variable. Each value based on 5 estimates.

Durations of the white stimulus in log-msec	*Threshold luminance of the red stimulus in log units*		
	Subjects		
	N.W.	*J.H.*	*C.H.*
1·6	–	–	–
1·7	1·725	1·615	1·575
1·8	1·800	1·715	1·680
1·9	1·905	1·755	1·757
2·0	1·960	1·840	1·815
2·1	1·927	1·845	1·860

occur, i.e. the original colour (red) persisted, no matter how much the intensity of this stimulus was reduced.

Fitting curves to the data. The data for the 2·0 log-mL white stimulus obtained with S.A. were made a standard, i.e. before any attempt was made to find curves fitting other sets of data, a smooth curve was fitted by eye to the points representing the results. After this, the same curve was used to fit other sets of points representing the data of S.A. that were obtained for the luminances of the white stimulus other than 2·0 log-mL, i.e. 1·8, 1·9, and 2·1 log-mL.

In retrospect, this attempt was quite successful and the curve fitted the points quite satisfactorily if allowances are made for minor deviations due to various experimental errors. (Fitting curves to the data of other subjects will be dealt with later.)

Description of the graphs. As can be seen from Fig. 5., the curves are spaced along the ordinate as a function of the luminance of the white stimulus, i.e. a curve is moved along the ordinate (upwards or downwards) according to the luminance of the white stimulus used. No obvious horizontal shifts of curves are seen with the change in the luminance of the white stimulus. Another outstanding feature of this graph is that the curves get shorter and shallower as the luminance of the white stimulus becomes lower. This is apparently due to increasingly greater portions of the curve disappearing from the left extreme ends (lower parts) of the curves. For example, the curve for the 2·1 log-mL stretches as far to the left as 1·55 log-msec on the abscissa. The one for the 1·8 log-mL, on the other hand, does not extend any further than 1·7 on the abscissa.

The dotted curve shown at the bottom of Fig. 5 is based on the data of an earlier study (Eysenck and Aiba, 1957). Each point in this curve repre-

sents the average of six readings obtained from a group of six subjects. The luminance of the white stimulus used was 1·7 log-mL (50 mL), though it must be pointed out that the conditions of this experiment were in some respect different from those of the present one, i.e. the natural pupil was used instead of the 3 mm artificial pupil and, moreover, the duration of the red stimulus was 20 msec instead of 4 msec as it was in the present study. The shape of this curve is again almost identical to the 2·0 log-mL one of the present study, and the curve would be best described as the one for the 2·0 mL white stimulus being displaced from its original position along the ordinate. Since there was the difference in the conditions as explained, it is rather fortuitous that this curve should be at the present position which would probably be occupied by the curve representing data for the white stimulus of 1·7 log-mL, had this luminance been actually tested in the present study, but it is significant that the shape of the curve is almost the same.

Differences among subjects. In Fig. 6 are shown curves based on the results of 3 subjects other than S.A., who were tested only with the white stimulus of 1·9 log-mL. The curve based on the results of S.A. tested under the white stimulus of the same luminance is also shown for comparison. The same curve which had been used for fitting data points in the preceding section, i.e. that of S.A. obtained with the 2·0 log-mL white stimulus, had again been tentatively used here for fitting the curves of the 3 subjects. There was a difference in the degree to which the points of each subject agreed with the standard curve of S.A., but they agreed with it better than with any other arbitrarily chosen ones. In this sense, applying the curve of S.A. to the results of any individual is thought to be justified. The graph shows that individual curves are spaced along the ordinate just as are the curves for various luminances of the white stimulus within an individual. There is no obvious displacement along the abscissa.

Analysis of the data in reference to Bunsen–Roscoe Law. From the results of the above study (using the subject S.A.) it is apparent that the "threshold" luminance of the red stimulus is a function of both the luminance and duration of the white stimulus, i.e. the threshold rises with an increase *either* in the luminance *or* in the duration of this stimulus, and lowered with a decrease in either of these variables. Having found that an increase (or a decrease) of the intensity has a similar effect as an increase (or a decrease) of duration, the question would arise whether these two variables are truly reciprocal, viz. whether the effect of the white stimulus remains the same regardless of any particular values of the luminance and duration of that stimulus, provided the total energy of light in the stimulus remains the same.

In order to find the answer to this question, the energy of light involved in the white stimulus (expressed by $i \times t$ where i is the luminance and t the duration), which was required to cause a "criterion effect", was plotted against the duration (t) in logarithmic scales. *The criterion effect* was defined as an occurrence of complete suppression of the red stimulus of a given luminance, when this was followed by white stimuli of various luminances and durations in the already described manner. Any level of luminance of the red stimulus may be chosen for this purpose, but a luminance of 1·88

(in our log unis) was selected, for it covered all 4 luminance levels of the white stimulus (see the dotted horizontal line in Fig. 2). The result of such plotting is shown in Fig. 7. As can be seen from it, $i \times t$ is approximately constant (3·74) up to about 1·85 log–msec. of t, but rises to the value of

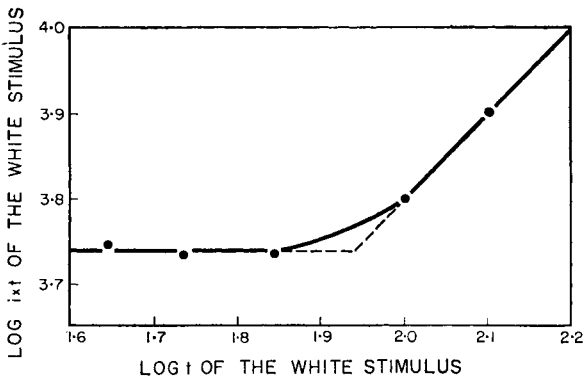


FIG. 7. The product of the luminance and the duration of the white stimulus ($i \times t$) that was required to produce the Bidwell phenomenon when presented with the red stimulus of a given luminance, plotted againts the duration of the former. Note that $i \times t$ is almost constant up to 1·85 log-msec and then on increases steadily. Subject: S.A.

3·8 at 2·0 log–msec. After this, $i \times t$ becomes strictly proportional to i. Unfortunately, the steps between luminances employed in the present study were not small enough for us to know how the transition occurs from the straight horizontal line to the oblique line which has a slope of +1. However, one would expect that where the curve is horizontal, temporal summation is complete and a true reciprocity between intensity and duration exists. On the other hand, the portion of the curve that is a line with the slope of +1 would mean that, in this range of stimulus values, increases in t no longer contribute to the increase in the *effectual* energy, and, therefore, cannot compensate for any possible reduction in i, i.e. there is no reciprocity. In other words, this is the region where the effect is entirely determined by the intensity of the stimulus, the duration having no measurable effect.

In order to see the relation between the energy involved in the white stimulus ($i \times t$) and the magnitude of suppressive effect of this stimulus upon the red stimulus, the threshold luminance of the latter was plotted against $i \times t$ of the former and a line is drawn through the data points. The result is shown in Fig. 8. It is noteworthy that the slope of the line is +1. There are slightly larger deviations of points at the lower part of the line. These, however, are quite small in comparison to the over-all trend in which all the points fall more or less exactly on the line in question, and they can be considered negligible.

There is one peculiar phenomenon which has been referred to already: the effect disappears, i.e. suppression of the red stimulus never occurs, no matter how low the luminance of the red stimulus is made, when t

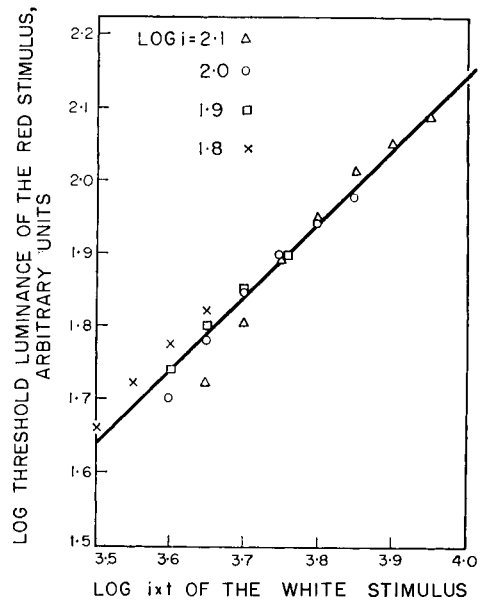


FIG. 8. The threshold luminance of the red stimulus as a function of the product of the luminance and the duration ($i \times t$) of the white stimulus. No points are plotted for thresholds which were obtained with the white stimulus exceeding 1·85 log–msec which is considered to be the upper limit of the duration for the complete temporal summation (see Fig. 10). The straight line drawn through the points is empirical.

of the white stimulus becomes smaller than a certain critical value. Moreover, this critical value of t varies according to the value of i of the white stimulus which happens to be used. In order to see this rather complicated relationship more clearly, this critical value of t was plotted against $i \times t$, in which i is the luminance of the white stimulus which was used to measure any particular critical duration. As can be seen from Fig. 9. in which the results are shown, the critical duration, t, is inversely related to the light energy involved, i.e. $i \times t$.

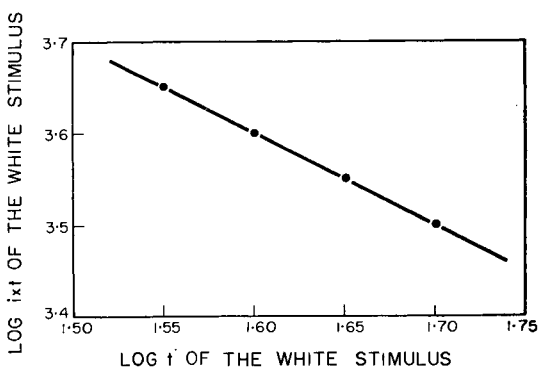


FIG. 9. The critical duration of the white stimulus below which the Bidwell phenomenon can never occur, plotted against the product of the luminance and duration ($i \times t$) of the white stimulus. Subject: S.A.

DISCUSSION

The effect of the white stimulus as determined by the "threshold" luminance of the stimulus was what one be expects red from the Bunsen–Roscoe law. This law, which was originally formulated to explain a purely physicochemical phenomenon, was first applied to sensory data in the field of threshold measurements (Bloch, 1885; Hartline, 1934; Graham and Margaria, 1935, and Karn, 1936). Since then, it has been tried on various psychophysical data and has been generally proved to hold true in spite of different situations in which they were obtained; it has been shown to hold for incremental threshold by Graham and Kemp (1938), Bouman (1952) Herrick (1956), Biersdorf (1955) and Barlow (1958) and also for acuity by Hunter and Stigler (1940). It is not surprising that we should have found the same effect with our phenomenon, inasmuch as it involves the two variables of a light stimulus – intensity and duration – like most other phenomena.

It is known that the range within which the Bunsen–Roscoe law holds varies widely from one function to another. It is also known that this range is dependent on many other factors, such as stimulus area, adaptation of the eye, the retinal locations stimulated, and so on. In the present study, its upper limit presumably lies somewhere between 1·85 log–msec (71 msec) and 2·0 log–msec (100 msec). As Graham and Margaria (1935) have pointed out, in human vision, the transition from the Bunsen–Roscoe region to the region where intensity becomes the sole determinant is never a sharp one as would be implied by the dotted line in Fig. 7. All previous studies with human subjects do in fact show that this transition is a very gradual one. If so, departure from the Bunsen–Roscoe relationship presumably occurs fairly early in this phenomenon, probably as soon as 1·83 log–msec, which is still in the region, is exceeded. The solid curve in Fig. 7. is based on this assumption.

As to the lower limit of the range, there were not enough higher luminances in this study to enable us to study much shorter durations. Previous studies do suggest, however, that the law holds for durations as short as, for example, $4{\cdot}1^{-3}$ msec (Brindley, 1952) when tested with the apparent brightness of a white stimulus. There is no reason why this should not hold true in our present experimental set-up also.

There is, however, an important proviso to the above statement arising from the fact that the phenomenon never occurs under certain values of $i \times t$ (however low the luminance of the red stimulus), if t becomes lower than certain critical values. As will be seen from Fig. 9., this critical value of t gets smaller and smaller with larger values of $i \times t$ used. This means that, in order to obtain the phenomenon with shorter durations of the white stimulus, $i \times t$ of this stimulus would have to be increased, in other words, its luminance would have to be increased accordingly (more than is needed to compensate the energy loss due to the shortening of the duration). To test the lower limit of the Bunsen–Roscoe range in this phenomenon an extremely intense light source would therefore be required.

It is believed that this rather peculiar effect which apparently results from the shortening of the duration of the white stimulus has a bearing

upon the implicit time (the latent period) of the response to the red stimulus in the visual system. It could be assumed that the effect disappears when the duration of the white stimulus becomes shorter than the implicit time of the response to the red stimulus, since, in such a state, the temporal sequence of the events necessary for the establishment of the effect may be reversed at the physiological level. Now we know from the works of Johnson and Bartlett (1956), Alpern and Faris (1956), Biersdorf (1958) as well as Bartley (1934), that the implicit time of responses in the visual cortex or in the retina (*b*-waves) is inversely proportional to the luminance of the stimulus involved. Thus, in our phenomena, if t (the duration) of the white stimulus is made smaller and smaller while i (the luminance) of it is kept constant, the threshold luminance of the red stimulus would be lowered correspondingly and this would, at the same time, lengthen the implicit time of the response to the red stimulus. Therefore, it is possible that under certain circumstances t of the white stimulus becomes shorter than the implicit time of the response to the red stimulus which is at or below its normally expected threshold. This would destroy the conditions favourable for the production of the phenomenon as explained above and the red stimulus would become visible. On the other hand, if $i \times t$ of the white stimulus is increased by an appropriate increase in i, the threshold luminance of the red stimulus gets higher and the implicit time of the response to this stimulus (at or near the threshold) would become shorter than the t of the white stimulus; hence the phenomenon reappears (the red becomes invisible) even though the duration of the white stimulus is still the same.

The curve based on the results of the subjects other than S.A. conform more or less to that of S.A., except that they are displaced from the latter's position along the ordinates. Judging from the detailed analysis of the family of curves belonging to S.A., this displacement may be attributed either to the variation in the effective luminance of the white stimulus, or, equally likely, to the variation in the effective luminance of the red stimulus. It is unlikely, however, that it is due to any change in the effective duration of the white stimulus, for, in this case, curves would be displaced along the abscissa. There is still another possibility that the displacement is caused by the change in the effective *duration* of the red stimulus. There is no way of finding out whether the change was in duration or in luminance. However, as our experimental variation of this stimulus was in the luminance and also, as the observed change is analogous to the change in its luminance, it would be better to refer to it as the change in the effective luminance of the red stimulus.

Finally, the implication of Fig. 8. may be considered. The straight line having a slope of $+1$ in this figure implies that, if we were to keep the red stimulus just visible (i.e. to keep it at its threshold) under various values of the white stimulus, we should increase or decrease the light energy involved in the red stimulus* in direct proportion to the values of the white stimulus. Thus, if a neutral density filter of varying densities is placed in

* As the duration of this stimulus is constant, we can talk of the amount of light energy involved in the stimulus in terms of the luminance.

front of the observer's eye and equal fractions of energy are subtracted from the two stimuli, there would be no change in the appearance of the red stimulus. It must be pointed out, however, that if the duration of the white stimulus is very short, the reduction of the luminance of *both* stimuli (whose durations are kept constant) would result in lengthening of the implicit time of the response to the red stimulus in excess of the actual duration of the white stimulus, as explained before, and because of this the phenomenon might disappear (the red become visible).

It will be remembered that Lehmann's (1952) method of determining the "threshold of the after-image" consisted in increasing or decreasing the luminances of both stimuli by means of a neutral filter whose density could be altered. It is possible that, in Lehmann's experiment it is the implicit time of the response to the coloured stimulus which largely determined the "threshold", for the duration of his white stimulus (as calculated on the basis of the speed of rotation of the disk and the area covered by the white sector) is as short as some of the critical durations of our white stimulus at which the Bidwell phenomenon disappeared. However, it is impossible to make comparison since the luminances of his white stimuli are not known to us.

THE EFFECTS OF ADAPTATION

(1) *Introduction*

The object of this study was to determine the effect of adaptation of the eye on the Bidwell phenomenon. More specifically, the effects of light and dark adaptation were studied to see how the curve, which was obtained in the previous study and which described the relationships between the duration of the white stimulus and the threshold luminance of the red stimulus, would be affected by various degrees of adaptation. The study was first done with one subject, and then the consistency of the results was tested with 6 others using a somewhat limited range of stimuli (this latter, using 6 subjects, will be referred to as the supplementary study, as opposed to the former which will be referred to as the main study).

(2) *Method*

The procedure in the main study was almost identical to the one described in the previous section. The only differing feature was that, instead of fixating at the 4·85 mL adapting field at the beginning of the measurements and between the flashes, the subject was adapted to a 93 mL field in the case of the light adaptation experiments and remained in complete darkness in the case of dark adaptation experiments. In both cases the subject had 15 min preliminary dark adaptation at the beginning of the experiment. The interval following each presentation of the stimulus sequence (the flashes) was considerably longer in the dark adaptation experiments than in the light adaptation experiments. This was to ensure that the dark adapted state of the eye was well maintained during the experimental sessions. Thus, the subject stayed in the dark for at least 10 sec during the interval

following the observation of the flashes in the former case. In the light adaptation experiments, the interval was the same as the earlier one, i.e., 5·3 sec. Light and dark adaptation experiments were performed on different days.

The procedure in the supplementary study was almost the same as in the main study, except that the subject was adapted to the 4·85 mL field as well. This moderate light-adaptation condition together with the light and dark adaptation conditions were all studied on a single occasion. The three conditions were experimented on in each subject in a balanced random order. The measurement was made twice for each condition and the average of these two taken as the final result. Ten min. dark adaptation preceded the measurements for whatever condition being studied.

In both the main and supplementary studies, only one luminance was used for the white stimulus, i.e. 2·0 log-mL. In the main study, the white stimulus was given at 11 different durations as in the preceding study. In the supplementary study, it was given at only one duration which was 1·7 log-msec. The duration of the red stimulus was, as before, kept at 4 msec.

(3) *Subjects*

The highly experienced subject (S.A.), who made most of the observations in the previous study, served as a subject again in the main study of the present series. The other 6 subjects used in the supplementary study were all research psychologists engaged in post-graduate research work at the Institute of Psychiatry, except for one who was a member of the secretarial staff (female) attached to the Institute. There were 4 males and 2 females. Their ages ranged between 21 and 34 with a mean of 27·5. None had gross anomalies in vision, and all had the acuity of 6/9 or better as tested by a Snellen chart.

(4) *Results*

(a) *The main study.* The results for both the dark and light adaptation conditions are given in Table 3. As a comparison, the readings obtained with the same subject in the previous experiment, in which the conditions were identical except for the adapting luminance (4·85 mL), are also given in the same table (this condition being designated as the "standard conditions"). The same data are presented in Fig. 10. (In the graph, the actual points representing the "standard conditions" are omitted for the sake of clarity, and just the curve which was drawn through them previously is reproduced.)

Following the method used in the previous section, the curve representing the "standard conditions" was fitted to the points representing the data for light and dark adaptations. It was found that the curve fitted well, but that it had to be moved along the abscissa, to the left, to fit the light adaptation points, and to the right, to fit the dark adaptation points. In the case of dark adaptation points, the curve had to be moved slightly downwards as well to attain the best fit.

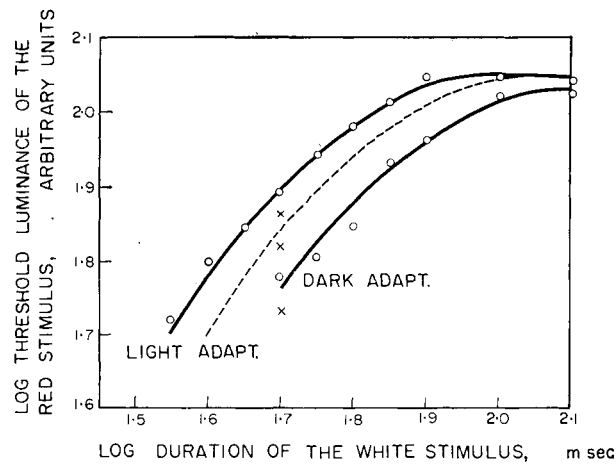


FIG. 10. Changes induced in the position of the curve describing the relationship between the duration of the white stimulus and the threshold luminance of the red stimulus following light and dark adaptation of the eye. Subject: S.A. The 3 crosses represent the average readings of the 6 subjects for (from top to bottom) light, 'standard' and dark adaptation conditions (see Fig. 11).

TABLE 3

Log threshold luminance of the red stimulus obtained with the "standard" white stimulus (luminance = 2·0 log–mL under the conditions of light and dark adaptation

Duration of the white stimulus in log–msec.	*Threshold luminance of the red stimulus in log units*		
	Dark Adaptation	*"Standard"*	*Light Adaptatton*
1·5	–	–	–
1·55	–	–	1·702
1·6	–	1·698	1·799
1·65	–	1·750	1·845
1·7	1·777	1·845	1·890
1·75	1·806	1·873	1·942
1·8	1·845	1·937	1·978
1·85	1·937	1·953	2·011
1·9	1·960	1·997	2·045
2·0	2·021	2·034	2·045
2·1	2·021	2·040	2·040

(b) *The supplementary study*. The threshold measures of the red stimulus in each subject under each condition are given in Table 4, and also presented graphically in Fig. 11. As can be seen from this, the thresholds under various degrees of light adaptation are consistent, in all subjects, with

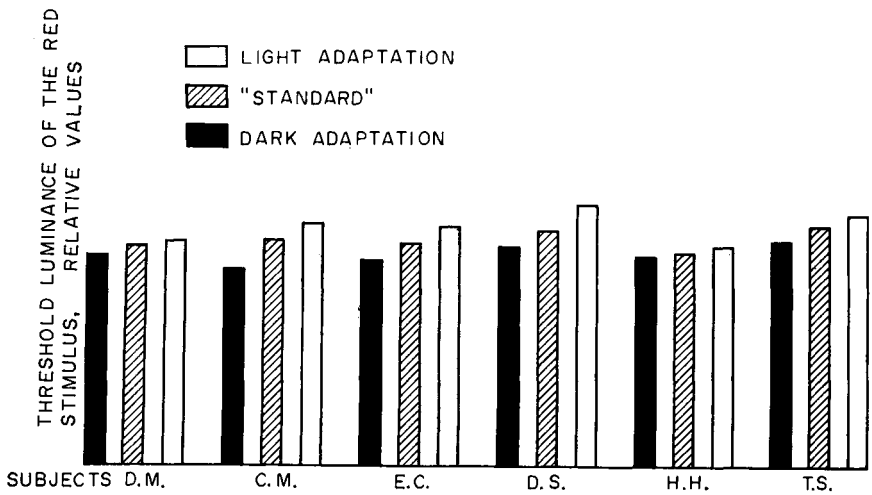


FIG. 11. The threshold luminance of the red stimulus for 6 different subjects under various states of adaptation. The duration of the white stimulus: 1·7 log–msec.

TABLE 4

Log threshold luminance of the red stimulus obtained from the six subjects under the "standard" as well as light and dark adaptation conditions (the white stimulus: luminance 2.0 log–mL and duration 1.7 log–msec).

Subjects	*Conditions*		
	Dark Adaptation	*"Standard"*	*Light Adaptation*
H.H.	1·725	1·740	1·770
D.S.	1·777	1·845	1·945
E.C.	1·705	1·785	1·845
C.M.	1·640	1·800	1·865
D.M.	1·725	1·770	1·795
T.S.	1·795	1·855	1·907
mean	1·730	1·820	1·860

the findings of the main study, i.e. highest for the light adaptation, lowest for the dark adaptation and in between the two for the moderate light adaptation.

The average of the threshold readings of the 6 subjects in each condition are shown as 3 crosses in the corresponding positions in Fig. 10. Making allowances for the fact that S.A. had rather high thresholds in the previous study as compared to other subjects (see Fig. 6.) it would be said that the agreement between the values of S. A. represented by the curves and the average values of the 6 subjects shown by the crosses is good.

DISCUSSION

We have seen in the previous section that there is a complete temporal summation in the effect of the white stimulus when the duration of this stimulus is within certain limits. There is no reason to suppose that complete temporal summation which had occurred in that study has not occurred in the present experiments since the experimental procedures were essentially the same in both cases and the basic shape of the curves describing the duration-threshold relationship had not altered by changing the adaptation condition. However, we found that the curve moves along the abscissa with changes in the adaptation condition, to the left under light adaptation and to the right under dark adaptation.

The above may be considered from the following two points of view: first, in terms of the point on the abscissa at which the curves start to flatten out (reach a plateau) and second, in terms of the threshold of the red stimulus under the two conditions at any point along the abscissa.

Let us consider the first point. As mentioned above, the curve for the light adaptation condition is displaced to the left along the abscissa. This would simply mean that the curve flattens out at a smaller value of t in the case of light adaptation than in the case of 'standard' condition. The opposite is the case with the dark adaptation condition. As the flattening out of the curves means that temporal summation in the effect of the white stimulus has ended in the regions of the duration concerned, the above mentioned observation can be interpreted as meaning that the upper limit of complete temporal summation occurs with smaller values of t (i.e. shorter durations) in the light adapted eye than in the dark adapted eye. This is in agreement with the results of a number of previous studies on the Bunsen–Roscoe law (Graham and Kemp, 1938; Ratoosh and Graham, 1951; Bouman, 1950; and Barlow, 1958).

Now the second point. It will be noticed, by inspecting Fig. 10, that the threshold for the red stimulus is always higher under the light adaptation than under the dark adaptation conditions, except at the highest value of t where the three curves virtually merge. This would mean that light adaptation increases the threshold of the red stimulus when t of the white stimulus is kept the same, or, to be more accurate, when $i \times t$ of the white stimulus is kept the same (since in the present experiment i is kept constant). Now, this is difficult to understand from the photo-chemical point of view, since the amount of photo-chemical substances available would be roughly the same for the two stimuli concerned (i.e. the red and the white) at any level of adaptation because the two stimuli were presented virtually simultaneously, and if so, the effects of adaptation would be analogous to placing

a neutral density filter of various values before the subject's eye (i.e.changing the intensities of both stimuli by an equal amount). This, we have seen,should not change the threshold of the red stimulus. This effect of adaptation becomes more meaningful, however, when we think of it in terms of neural mechanisms involved in the suppression of the red stimulus. For example, if the suppression is caused by pre-excitatory inhibition as Eysenck and Aiba (1957) have earlier suggested or mediated by such mechanism as lateral inhibition which Barlow, Fitz Hugh and Kuffler (1957) have demonstrated in the cat's eye, then the threshold of the red stimulus would become higher in the light adapted than in the dark adapted eye, for both types of inhibition are shown to increase with the increasing level of light adaptation.

THE DRUG STUDIES: TIME COURSE OF EFFECTS

(1) *Introduction*

The experiments reported in the preceding sections have revealed that there exist rather simple and straightforward relationships between the intensity of the white stimulus and the threshold luminance of the red stimulus. Of particular importance would be the luminance–duration relationship of the white stimulus, which was based on the finding of various combinations of the luminance and duration of this stimulus needed to suppress the red stimulus of a given intensity. We have seen that this relationship is explainable in terms of the Bunsen–Roscoe law. This might be taken to mean that the magnitude of the suppressive effect of the white stimulus is, to a large extent, directly determined by the amount of initial photo-chemical bleaching in the receptor which was caused by the white stimulus. Though strongly suggesting a photo-chemical connection, this does not, however, explain the whole process responsible for the occurrence of the Bidwell phenomenon. Indeed, even the simplest of all visual functions, absolute threshold of light, cannot be fully understood without the knowledge of neural elements involved in it. A greater part must be played by the neural mechanisms, when the phenomenon becomes as complex as the one with which we are dealing. How, for instance, could one explain suppression of the red stimulus by the succeeding white stimulus in terms of pure photo-chemical processes? No known photo-chemical theory can account for such an occurrence. In this sense, our phenomenon belongs to the same category of phenomena as Crawford's effect (Crawford, 1947) in which thresholds for a test flash are raised by a light-adapting field when the former *precedes* the latter by a few milliseconds. Likewise, the rise of thresholds of the red stimulus shown in our light adaptation experiments cannot be due to a photo-chemical change but must be due to a more complex neural reorganization of the retina such as those suggested in the discussion of the results.

To know that the neural factors are important determinants of the phenomenon is one thing, and to know the exact nature of such neural mechanisms is another. Even a detailed electro-physiological study on the human subjects in precisely the same conditions as in the present experi-

ments may not resolve this matter, so long as it relies on inferences relating electrical activities in the nervous system to something we actually perceive as a sensory experience. However, this does not prevent us from investigating the various determinants of a phenomenon and speculating concerning the possible physiological mechanisms underlying it on the basis of observed facts. To help us in considering the mechanism, there is, fortunately, quite a large body of work in the field of visual physiology. Inasmuch as most of these physiological studies are concerned with the relationship between various stimulus variables and the physiological responses that have arisen from stimulation, they may be said to provide at least some common ground with psychologists whose main task is also to establish relationships between stimulus variables and responses. Thus, it is no more improper for a psychologist to speculate about his own findings in terms of what physiologists have found in a similar situation, than for a physiologist to speculate about his results in terms of what psychologists have found in a psycho-physical situation. We could thus enumerate any number of physiological processes that are likely to be operative in a situation like the present one. This, however, is only the starting point. We must go on to find, by proper experimentation and by the process of elimination, which of these processes is the one most likely to be responsible for the particular psychological phenomenon in question. There could be many forms of such experimentation. We believe that drug studies are just one form of experiment aimed at this objective.

In general, drug studies are of value in the dual sense. On the one hand, they might help to throw light on the mechanism or the process that is involved in any particular sensory experience; this is so when we know already the site and the action of drugs. On the other hand, if we possess some knowledge of the physiological processes underlying our psychological phenomena, then we can make use of this knowledge to find out the site and the action of drugs. The drug experiments to be reported in the following sections have both of these characteristics. This is due to the rather sad fact that we know neither the mechanism responsible for our sensation nor the exact mode of action of the drugs. Under these circumstances, the best we can do is to combine what little information we have on the two topics and to make it into a hypothetico-deductive system that is at least coherent and intelligible. This may look a circuitous sort of proof, but our aim at the present stage is not to prove anything; rather it is to accumulate facts and to interpret them as we go on so that we can have a working hypothesis for further investigation.

(2) *The Experiment*

This experiment was undertaken to test the effects of amylobarbitone and dexamphetamine upon the threshold luminance of the red stimulus when the latter was followed by a white stimulus of a given luminance. In this experiment, a rather long duration was used for the presentation of the white stimulus (2·54 log–msec) and there was no temporal summation for this stimulus. The reason for doing this was that one could thereby elimi-

nate effects of drugs on the temporal summation for the white stimulus which might otherwise contaminate the results. The experiment was also designed to yield information as to the exact time from the administration at which the effects of the drugs would start to be active. (A preliminary account has been given in Aiba, 1960.)

(3) *Method*

Stimuli – The apparatus used for this experiment was identical with that used in the preceding psycho-physical experiments. However, for this particular study, it was set to give the following sequence of stimuli: (1) a dark interval lasting approximately 3·5 sec; (2) a red stimulus, surrounded by a white annulus, lasting 10 msec; and (3) a white stimulus lasting 350 msec (2·54 log–msec) immediately following the red stimulus. The luminances of the white stimulus and the white annulus surrounding the red stimulus were both fixed at 95 mL which is about the same as the "standard" luminance (2 log–mL) of the psycho-physical experiments. Other features of the stimuli unmentioned here were identical to those which occurred in the preceding psycho-physical experiments.

Subjects – Five subjects were used in the present experiment, all of whom were students in a post-graduate course in clinical psychology. The ages of the subjects ranged between 21 and 33, with a mean of 24. None had any gross defect in vision and all had a visual acuity of 6/9 or better as tested by a Snellen chart. There were 3 males and 2 females.

Procedure – After 15 min of dark adaptation, the subjects were told to look at the stimulus patch through the 3 mm artificial pupil and to increase the luminance of the red stimulus by turning the knob of the variac, until they could see the first suggestion of red in it. The subjects were allowed to repeat this if they were in doubt on the first occasion. (Strictly speaking, the eyes of different subjects would be light-adapted to slightly different extents because of this procedure. However, experimental errors resulting from this were expected to be quite small, since it had been shown in the preceding study that the state of adaptation has little influence on the threshold when the duration of the white stimulus is relatively long.) As soon as they were satisfied with their adjustment, they resumed their dark adaptation by staying in the dark until the next trial began. Four of these trials made up one session, and during the next 2 hours, the same procedure was repeated every 20 min. Thus, there were altogether six sessions, each consisting of four trials. Between trials, there was an interval of 2 min, and between the fourth trial and the first trial of the next session there was an interval of 5–7 min, depending on how quickly the subjects made each adjustment. During these intervals also they stayed in the dark. Owing to the great difficulty experienced by the subjects in keeping the eye close to the eye-piece and in maintaining the right fixation during the lengthy course of the experiment, a certain amount of prior training was necessary. This consisted of repeating part of the experimental procedures, i.e. the first, the second and the third sessions, on three different days prior to the experiment proper; consequently, the subjects were

thoroughly familiarized with the experimental procedure when they entered the experiment.

Drugs – The drugs were administered between the second and the third sessions (usually immediately after the second session) each day. The drugs used and their doses were identical with those used in the Eysenck and Aiba (1957) studies, (1) dexamphetamine sulphate (Dexedrine, as supplied by Smith Kline and French) 10 mg; (2) amylobarbitone sodium (Sodium Amytal, as supplied by Lilly) 290 mg; and (3) the placebo. These were given in identically appearing capsules and taken orally. Each subject was given the drugs in random order over the three days of testing. Between experimental days at least a week elapsed, so the sequence effect due to the residual drug effects was negligible.

(4) *Results*

An analysis of variance was applied to the data of the two pre-drug sessions, Sessions I and II, to see the general trend of the readings prior to the administration of the drugs. This analysis disclosed that there was a large day-to-day variation in the basic levels of threshold readings for each subject and also that there was no systematic trend in that variation. Consequently, the analyses of the post-drug sessions were based on the *relative* values of the readings within each day. For this purpose, the average of the four readings of Session I was chosen as a reference for that particular day, and this was subtracted from readings of all subsequent sessions of that day. Only the average of the four trials in each session was used for the analyses.

The outcome of the final analysis of variance is shown in Table 5.

TABLE 5

Analysis of variance for the results of the post-drug sessions (Sessions II—VI).

Source	*D.F.*	*S.S.*	*M.S.*	*V.R.*
Between People	4	12 206	3 051·5	25·39*
Between Sessions	4	5 482	1 370·5	11·40*
Between Drugs	2	2 451	1 225·5	10·20*
Interaction				
Between Sessions/People	16	2 834	177·1	5·35*
Between Drugs/Session	8	2 022	252·8	7·63*
Between People/Drugs	8	1 776	222·0	6·71*
Residuals	32	1 059	33·1	
Total	74	27 830		

* Significant at 1% level

As can be seen from this, the F tests based on all sources of variation are at a highly significant level. Of particular importance would be the variations due to drugs and those due to the interaction between drugs and sessions. These show that the drugs produced definite effects, but that the time at which the effects of the drugs became manifest was not the same. How each drug altered the threshold in course of time can seen from Fig. 12.

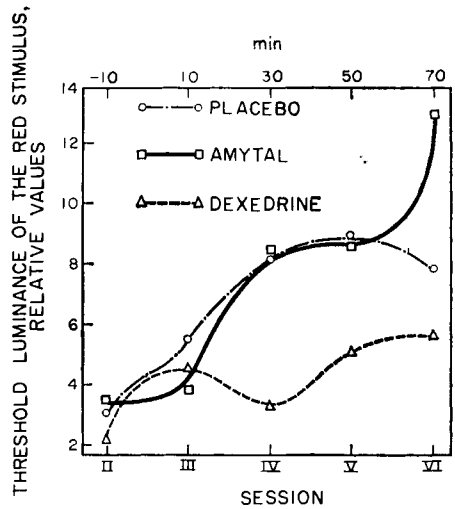


FIG. 12. A graph showing the threshold luminance of the red stimulus at various times from the administration of the drugs. The points are freely fitted with smooth curves. The drugs used: Amytal (amylobarbitone sodium) and Dexedrine (dexamphetamine sulphate). The duration of the white stimulus: 2·54 log–msec. The number of subjects: 5.

The significances of the differences between average readings for each session under different treatments were tested using *T*-tests. It was found that there are no significant differences between the treatments either in Session II or in Session III. In both Sessions IV and V, however, there is a significant difference between the placebo and dexamphetamine (Session IV: $t = 5{\cdot}277$, $P < 0{\cdot}01$; Session V: $t = 4{\cdot}177$, $P < 0{\cdot}01$) and also between amylobarbitone and dexamphetamine (Session IV: $t = 5{\cdot}469$, $P < 0{\cdot}01$; Session V: $t = 3{\cdot}793$, $P < 0{\cdot}01$), but there was no significant difference between the placebo and amylobarbitone in either of these sessions. In Sessions VI, the last session, the difference between amylobarbitone and the placebo becomes significant ($t = 5{\cdot}606$, $P < 0{\cdot}01$) and the differences between dexamphetamine and the placebo ($t = 2{\cdot}473$, $P < 0{\cdot}05$) and between dexamphetamine and amylobarbitone ($t = 8{\cdot}080$, $P < 0{\cdot}01$) still remain significant. As to the direction of the effects of the two drugs, they were in the opposite direction, i.e. *dexamphetamine lowered the threshold* while *amylobarbitone raised it*, as shown in Fig. 12.

We found in this study that the effect of dexamphetamine and amylobarbitone on the threshold of the red stimulus are opposite, i.e. the former lowering it and the latter increasing it.

We also obtained information concerning the latent period of the effects of the drugs on Bidwell's phenomenon. When the above reported results are considered in terms of the time from the administration of the drugs we find that the threshold was lowered by dexamphetamine within 30 min after the administration of the drug and it remained so for the rest of the testing time (approximately 40 min from when it first became apparent); on the other hand, with amylobarbitone, the effect did not appear till about 70 min after the administration. These two results agree fairly well with those obtained in a study dealing with the effects of drugs on critical frequency of flicker (Roback and others, 1952). There is one unexpected effect which may deserve a comment. As will be seen from Fig. 12, the thresholds were generally increased, under all treatments, toward the end of the experimental session. A possible explanation for this is that it was caused by gradual light adaptation of the eye during the testing sessions. Though dark adaptation periods were inserted between testing sessions, these may not have been long enough for the eye to recover fully from the partially light adapted states caused during preceding testing sessions and the eye may have been progressively light adapted in the course of the experiment. It is true that the effect of adaptation on the threshold has been shown to be quite small particularly when the duration of the white stimulus is long. Nevertheless, it may be as substantial as the effect of the drugs. More stringent control of the state of adaptation of the eye, therefore, might be indicated if we were to compare the effects of drugs with those of adaptation. On the other hand, the results of the present experiment are considered quite valid since we are here comparing the placebo condition with the two drug conditions, among which states of adaptation could be considered equal. However, large individual differences and day-to-day variations in thresholds, observed in the present study, might be accounted for by fluctuation in states of adaptation; it will be remembered that the luminance–duration curve was slightly shifted downwards under the dark adaptation condition (Fig. 10), suggesting the possible effect of dark adaptation.

Finally, we should briefly consider the possible mechanism through which the effects of drugs are mediated to cause changes in the threshold of the red stimulus. Since a long duration was used for the white stimulus in the present study, a loss in the temporal summation, if any, should have been well compensated. The results, therefore, must be due to some other mechanism. Our psychophysical studies showed that, when a white stimulus as long as the one used in the present experiment is employed, only variations in its luminance can produce any noticeable change in the threshold of the red stimulus. It must be assumed from this that the effects of drugs consisted in altering the effective *luminance* of the white stimulus. It is true, this statement offers only a description of the results, but it is possible that some drugs in fact have this effect, and, furthermore, it is highly probable that amylobarbitone is one of them. Both Danis (1956, 1959) and Arden and others (1960) found that barbiturates increased the electrical responses

of the eye (i.e. the component responses the electroretinogram) of cats, rats and rabbits to single flashes in the same way as actual increases in the luminance of the stimuli (amplification of the responses). This effect was more marked with comparatively bright light and when the eyes were dark adapted. So the white stimulus used in our experiment might have had a stronger inhibitory effect on the red stimulus when it was presented to the subjects who had been given amylobarbitone.

As regards dexamphetamine, there is as yet no study on the effect of this drug on the electrical response of the eye. Therefore, there is no ground on which we can restate our previous argument.

THE DRUG STUDIES: UNDER VARYING DURATION OF THE WHITE STIMULUS

(1) *Introduction*

We have seen in the preceding section that the threshold of the red stimulus was raised by amylobarbitone and lowered by dexamphetamine when white stimulus that followed the red was 350 msec (2·54 log–msec). However, it is possible, as was adumbrated in the preceding section, that amylobarbitone, as well as having a threshold raising effect, might also have an inhibitory effect on the temporal summation of the effect of the white stimulus, reducing its extent. If so, under amylobarbitone, the white stimulus of duration t may become like a white stimulus of duration $t–a$, a being the decrement in the effective duration produced by the drug, and hence, the cancellation of its threshold raising effect (if not the lowering of the threshold) would result. However, this would occur only when the duration of the white stimulus is still within the limit of temporal summation.*

To test the validity of the hypothesis, that amylobarbitone reduces the temporal summation, we must obtain curves describing the relationship between the threshold of the red stimulus and the duration of the white stimulus (similar to those obtained in our psycho-physical studies) with the subject who is subjected to the influence of the drug. In fact, an attempt similar to this has already been made in the Eysenck and Aiba experiment in conjunction with the study which has been referred to and the results suggested that the above mentioned effect might indeed exist. Unfortunately, their data for the drug conditions, which consisted of ingestion of dexamphetamine and amylobarbitone, may be contaminated by variations in pupil size, etc. (though it must be admitted that their data for the placebo condition appear to correspond reasonably well to our psycho-physical data). It was, therefore, thought worth-while to re-examine the effect under more controlled conditions before speculating about this particular aspect of the effects of drugs. Thus, the purpose of this experiment may be expressed as a re-examination of the changes of the effects of drugs on the threshold of the red stimulus

* It is very unlikely that the temporal summation should be increased when there is already a complete summation, since in this state the energy of light is used most effectively and a further increase in summation is unthinkable. On the other hand, the decrease in summation could occur at any level of summation.

following changes in the duration of the white stimulus, which had been attempted by Eysenck and Aiba (1957).

The study was divided into two parts, one conducted in the same way as the psycho-physical study, and the other under somewhat simplified conditions (especially in respect to procedure). One well-trained subject took part in the first part of the study, and six untrained subjects took part in the second part; the two parts of the study were intended to complement each other.

(2) *Experiments*

(a) *Experiment with one experienced subject*

Method – The stimulus conditions were identical to the "standard" conditions of the psychophysical study, i.e. the white stimulus was unchanged in respect to its luminance which was fixed at 2·0 log–mL but was varied in duration between 1·6 and 2·1 log–msec. The duration of the red stimulus, whose luminance was the dependent variable in the experiment, was kept at 4 msec throughout. The adaptation level was also the same as that of the psycho-physical study, i.e. 4·85 log–mL. The procedure, too, was the same as that used in the psycho-physical study, except that the subject took drugs before undertaking the test. To assess the non-drug condition (equivalent to the placebo condition), the thresholds of the red stimulus were also measured before the administration of drugs using the 1·7 and 2·1 log-msec white stimulus.

Special precautions were taken to complete the experiments while the effects of drugs were more or less at a stable level. The measurement was started as soon as the estimated latent period of the effects of each drug (see the preceding section) had passed. Judging from the threshold changes occurring under dexamphetamine, it was thought that the effect of the drugs would remain more or less at the same level for at least 40 min after it had first become apparent. (As for amylobarbitone, there are no data on the duration of the effect, but there is no reason to suppose that this drug has a much faster rate of dissipation from the site of its action than dexamphetamine.) Furthermore, measurements were carried out in deliberately randomized order, i.e. instead of starting the test always from the shortest white stimulus and finishing it with the longest (as was the case in the psycho-physical study), the long and short durations were intermingled randomly. Thus, if there were any unevenness of the effects of drugs, these should show up in the irregularity of the curve.

The same well-trained subject who made most observations in the psycho-physical study served as a subject in the present experiment again. The drugs used and their respective doses were identical with those used in the preceding study, i.e. dexamphetamine 10 mg and amylobarbitone sodium 290 mg. There was no placebo given in this study and the subject knew which drug he was given on each occasion. He was, however, ignorant of the results of the experiment till it ended.

Results – The results are shown in Table 6 and Fig. 13. The latter requires some explanation. To draw this figure, the curve that had been fitted to

the data representing the "standard" condition of the psycho-physical study was applied again to fit the points representing, respectively, each drug and the "no drug" conditions. The fit was fairly satisfactory. Thus, we now have two pairs of curves, each one consisting of curves representing the drug and the "no drug" conditions. The graph in Fig. 13 was obtained by moving one of the pair of curves so that the curves representing the "no

TABLE 6

Log threshold luminance of the red stimulus under amylobarbitone and dexamphetamine. The white stimulus: luminance 2·10 log–mL; duration varied between 1·6 and 2·1 log–msec. Figures in brackets show corresponding values for "no drug" conditions

Duration of the white stimulus in log–msec.	*Threshold luminance of the red stimulus in log units.*	
	amylobarbitone	*dexamphetamine*
1·6	1·775	1·656
1·7	1·855 (1·800)	1·837 (1·857)
1·8	1·925	1·900
1·9	2·030	1·970
2·0	2·075	2·000
2·1	2·090 (2·015)	2·004 (2·029)

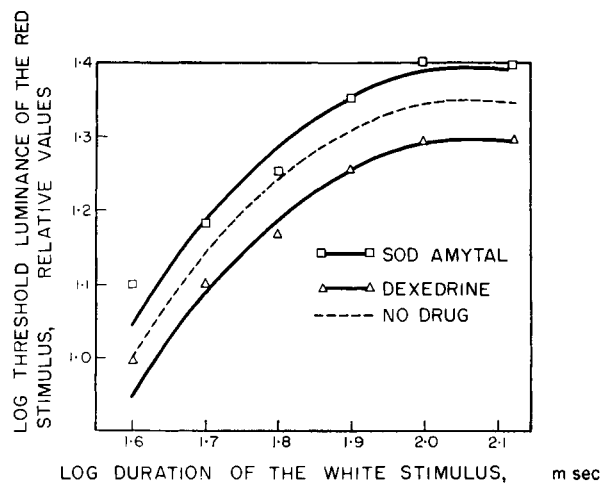


FIG. 13. A graph showing the effects of drugs on the threshold luminance of the red stimulus measured with various durations of the white stimulus.

drug" conditions in both pairs were joined together. On account of this procedure, the ordinates are given in relative values.

The graph shows that there is no substantial change in the effects of the drugs as the duration of the white stimulus was varied from 1·6 to 2·1 log-msec; none of the points representing either one of the drugs crosses to the other side of the "no drug" curve. The curves are thus parallel to each other, rather like those representing the threshold luminance of the red stimulus under various luminances of the white stimulus (Fig. 5.)

(b) *Experiment with 6 untrained subjects*

Method – The stimulus conditions of the experiment reported here were virtually the same as those described earlier (a), except that, in the present experiment, the durations of the white stimulus were limited to the following three: 1·6, 1·8 and 2·0 log–msec. As all subjects used in this study were inexperienced in this type of experiments, a certain amount of training was needed prior to the experiments proper. The training consisted of actually making judgment of the colour of the stimulus (reporting whether it was completely green or not) in the same way as in the real experimental sessions. After a little training, most subjects were able to give fairly reliable results. When the subjects were judged to have had sufficient training, the actual experimental sessions were begun immediately.

The experimental procedure was the same as that which occurred in the psycho-physical study. However, the length of the experiment was considerably reduced owing to the smaller number of conditions explored in this study. None of the subjects were psychologists, and, as pointed out earlier, none had previously taken part in any psychological experiment as a subject. There were 4 males and 2 females, their ages ranging between 25 and 45 with a mean of 32. They were all paid volunteers. None had gross anomalies in vision and all had acuity of 6/9 or better (Snellen chart). The two drugs used in the experiment reported in (a) and a placebo were used in the present study. Their respective doses, the time between the taking of each drug and the start of the experimental session were also the same as before. The two drugs and the placebo were given to each subject in the balanced random order over the 3 days of testing. Neither the subject nor the experimenter knew which drug was given to the subject on each occasion (the double blind technique).

Results – The threshold reading for each subject under the two drug and the placebo conditions are shown in Table 7. As can be seen, the thresholds of the red stimulus under amylobarbitone are consistently higher than those under any other condition, whereas those under dexamphetamine are consistently lower. To test the statistical significance of these results, an analysis of variance was carried out. The outcome of this analysis is shown in Table 8, from which it will be seen that the changes of the threshold produced by the drugs are significant, but that this effect is independent of the duration of the white stimulus – the interaction between Treatment and Duration being insignificant. The differences between treatments

TABLE 7

Thresholds of the red stimulus under amylobarbitone, dexamphetamine and a placebo for three different durations of the white stimulus

		Subjects						
Duration	*Treat.*	*C.F.*	*M.Q.*	*J.H.*	*C.H.*	*J.M.*	*N.W.*	*Mean*
1·6 log–msec.	Amy.	62·5	81·5	67·6	62·2	66·5	72·5	68·8
	Pla.	67·5	77·5	64·2	61·6	65·5	66·5	67·1
	Dex.	68·5	70·5	63·4	61·0	62·5	64·5	65·1
1·8 log–msec.	Amy.	76.5	87.5	75.4	69.0	76.5	73.5	76.4
	Pla.	75·5	86·5	73·2	71·0	72·5	73·5	75·4
	Dex.	78·5	81·5	69·8	70·2	76·5	69·5	74·3
2·0 log–msec.	Amy.	97·5	107·5	84·8	84·0	91·5	97·5	93·8
	Pla.	88·5	100·5	83·0	81·0	85·5	100·5	89·8
	Dex.	89·5	96·5	81·8	81·8	87·5	81·5	86·4
Mean		78·3	87·7	73·7	71·3	76·1	77·7	

(Each value is a mean of 4 estimates of the threshold; units used are the same as in Table 10.)

TABLE 8

Outcome of analysis of variance of the effects of amylobarbitone and dexamphetamine upon the threshold of the red stimulus

Source	*D.F.*	*S.S.*	*M.S.*	*V.R.*
Between Treatments	2 (2)	17·337 (66·408)	8·669 (33·204)	7·337** (5·826)*
Between People	5 (5)	144·048 (409·261)	28·810 (81·852)	24·386*** (14·363)**
Between Duration	2	488·886	244·443	206·910***
Interaction between Treatment and Duration	4	4·454	1·114	0·943
Residuals	40 (10)	47·254 (56·993)	1.181 (5·699)	
Total	53 (17)	701·198 (532,662)		

(* significant at 5% level
** „ at 1% level
*** „ at 0.1% level)

(In the brackets is shown outcome of analysis for which the three durations are combined. Note that the treatments are still significant.)

are not significant when any one of the three durations is taken singly, (when tested by T-tests making use of the residual variance) except in the case of the difference between the dexamphetamine and amylobarbitone conditions for the longest duration used (2·0 log-msec). This failure to discriminate might be due to the rather large error variance. However, they are all in the same direction as might be expected from the insignificant interaction between Treatment and Duration which has already been mentioned and they support the statement that the effects of drugs are independent of the duration of the white stimulus.

DISCUSSION

The changes that occurred in the threshold reading under the two drugs are analogous to those produced by varying the luminance of the white stimulus. Thus, the shape of the curve, describing the relationship between the duration of the white stimulus of a constant luminance (say, 2·0 log-mL) and the threshold luminance of the red stimulus, was essentially unchanged but its position along the ordinate was altered – in amylobarbitone it was shifted upwards and in dexamphetamine in the opposite direction. This is in contradiction to Eysenck and Aiba's (1957) earlier finding that both dexamphetamine and amylobarbitone lower the threshold of the red stimulus.

However, we could say that the control of the experimental conditions in the study reported here is, on the whole, much more satisfactory and adequately safeguards against various complications to which Eysenck and Aiba's study might have been subjected. Therefore, the effects of drugs on the threshold found in this study could be regarded as more genuine.

If we are satisfied that the results of the present experiment are genuine, what would be their implications? The argument that the drugs affect the effective luminance of the white stimulus (see 'Discussion' of the preceding section) still holds true after the present study and is indeed even more cogent since we have found in this study that the shortening of the duration did not change the basic feature of the effects of drugs. Indeed, if the drugs affect the effective *duration* of the white stimulus, we would have found the effects being manifest only when the duration is still within the limit of temporal summation. Conversely, if the effects of drugs are obtained only when the duration of the white stimulus is long (as was the case in the preceding study) the effects would have been gone when the duration of the white stimulus was reduced. As neither was found to be the case, and the effects appeared to be fairly constant over the whole range of duration tested, as if they were caused by actual changes in the luminance of the white stimulus (see Fig. 13), the effects can justifiably be considered as changes in the effective luminance of the white stimulus. We have seen, in the preceding section, that the drugs might indeed be capable of producing such changes. However, the problem still remains how the changes in the luminance of the white stimulus (whether effective or real) can alter the thres-

hold of the red stimulus. This, of course, is related to the more basic question of what causes the Bidwell phenomenon in the first place.

The mechanisms underlying the Bidwell phenomenon are not very clear, partly because we do not yet know the precise nature of the phenomenon which we are investigating (the present series of studies being one attempt to discover this), and partly because there have not yet been many directly relevant physiological studies. Pre-excitatory inhibition, however, is one of the few possible intervening mechanisms that could be suggested (Eysenck and Aiba, 1957). According to this hypothesis, pre-excitatory inhibition caused by the white stimulus inhibits the excitatory potentials caused by the red stimulus, because both processes are made to occur simultaneously by the particular time sequence in which the two stimuli were presented.

Now there is some ground to assume that the a-wave of the electroretinogram is an observable manifestation of pre-excitatory inhibition (Granit, 1947). If so, this inhibition is either increased or decreased by varying the intensities of a stimulus (Granit, 1947; Alpern and Faris, 1956; Biersdorf, 1958), and this fits in well with our observation that the drugs alter the effective luminance of the white stimulus.

One difficulty arising in connection with an argument like the above one is that the drugs may increase (or decrease) the excitatory potential caused by the red stimulus as well (it may be remembered that barbiturates enhanced all electroretinogram responses, not just the a-wave). If this is the case, the effect of amylobarbitone may be likened to the reduction of the density of an optical filter which has been inserted between the stimulus and the observer's eye, with the resultant increase in the intensities of the two stimuli. Now, inspection of our Fig. 8. clearly shows that adding equal fractions of energy to both stimuli by reducing the density of the filter would not change the appearance of the phenomenon since the data point would simply move along the +1 line; hence the threshold would remain exactly the same.

The above mentioned difficulty would remain insuperable so long as the process responsible for the production of the Bidwell phenomenon is pictured as the b-wave caused by the red stimulus being inhibited by the a-wave caused by the succeeding white stimulus. The following hypothesis may be put forward as an alternative. The two stimuli, the red and the succeeding white, instead of giving two separate responses, might give a summated response because the two stimuli are so close in succession. The red is suppressed, thus, because it is given in the beginning of this continuous sequence of stimuli. If so, we could ignore the b-wave part of this unified response, whether it is affected by the drug or not, and consider only the a-wave part of it, which would be increased by amylobarbitone and consequently cause the threshold of the red stimulus to rise. In this explanation, the results of the drug experiments are simply interpreted as due to an increase (under amylobarbitone) or a decrease (under dexamphetamine) of the a-wave (or pre-excitatory inhibition).

It is to be admitted that it is still premature to speculate at this juncture. Indeed the interpretation of the results would be quite different according to the locus of the (neural) interaction which causes the Bidwell phenomenon.

It could be in centres higher up in the visual pathways, or it may be in the retina itself. Even when proved to be in the retina (which the results of our experiments on dark and light adaptation seem to indicate) there still remains the problem of the layer of the retina affected. Only further experimentation can prove the correctness of any particular hypothesis.

THE DRUG STUDIES: COMPARISON WITH C.F.F.

(1) *Introduction*

So far we have been considering the Bidwell phenomenon only in relation to the two successive stimuli involved, i.e. the red and the succeeding white. It must be remembered, however, that when the red stimulus is presented, it is never presented without the white surround. This white area seems rather important, since the complete suppression of the red never occurs without it. It would be interesting to see to what extent this white area is involved in determining the threshold of the red under the drugs. Indeed, if the effects of drugs are specifically on the interaction caused by this white surround and the red stimulus, then the kind of explanation advanced in the preceding sections would become less tenable since it is all based on the assumption concerning the functioning of the two *successive* stimuli.

The test, which would be able to determine how much of the effect of drugs is due to influences on the functioning of the white surrounding area, must be a test that can single out from sundry effects the drugs might possess, those that are specifically on lateral interactions in the visual system.

Unfortunately, we do not know of any conventional visual test that would satisfy the above requirement; nearly all tests are measures of a conglomeration of processes most of which are quite unknown to us. Therefore, we have to construct an experimental situation where we could single out a certain aspect of the effect of drugs by suitably manipulating the experimental variables, though the test used in the experiment may not be itself capable of such differentiation. This, we believe, is realized in a situation where c.f.f. is measured under various surround illumination.

Generally, the effect of drugs on c.f.f. as such could be accounted for by any one of the following three hypotheses, i.e. the pre-excitatory inhibition hypothesis (as in Granit, 1947), the filter factor hypothesis (as in Granger, 1959), and the recovery period hypothesis (see Aiba, 1959). Therefore, c.f.f, by itself may be of little use for the purpose of determining the presence or absence of lateral effects of drugs. However, the effect of surround illumination on c.f.f. may be thought of as a different category of tests altogether and might be profitably employed for the above purpose. To be more precise, as the effect of surround illumination on c.f.f. is most likely to be based on lateral interaction in the visual system (see Aiba, 1959), by seeing how this c.f.f. surround effect is altered by drugs, we could at least test the compatibility of the hypothesis that the observed effects of drugs on the Bidwell phenomenon is due to the drug's action on the lateral interaction between the red stimulus and the white surrounding area.

In terms of the actual experiments to be carried out, our hypothesis is as follows: when the surround luminance is equal to or just below the test field luminance and both fields are above photopic levels of luminance, the c.f.f. would be raised by a drug that increases lateral inhibitory interaction from the more pronounced darkness of the dark phase of the flicker due to enhanced contrast. On the other hand, the same drug should lower the c.f.f. when the surround luminance is higher than the test field luminance, because it would then be mainly the light phase of the flicker that suffers depression. Furthermore, on the basis of the assumption that the threshold of the red stimulus in the Bidwell phenomenon is largely determined by the lateral interaction involving the white surround, it is the drug which increases the threshold of the red stimulus, i.e. amylobarbitone that should alter the c.f.f. in the way just described.* By the same reasoning, dexamphetamine should have the opposite effects, i.e. should lower c.f.f. under the equal surround and raise it when the surround is brighter. A more detailed discussion of the points is given in Aiba (1959) which also contains a preliminary account of the results of the experiment to be reported.

(2) *Methods*

The apparatus used has already been described (see 'Apparatus'). The stimulus used consisted of a circular flickering source, centred on an illuminated circular surrounding field. The stimulus, together with the surrounding field, subtended a visual angle of 5°, while the flickering source itself subtended a visual angle of 40′. The two levels of luminance employed for the surrounding field were 2·4 and 94·2 mL, respectively. The colour temperature of both surrounds was approximately 3500° K. The flickering source which was red–orange in colour having neon light spectrum had an apparent brightness of 2·9 mL when flickering just above fusion. The subject viewed the flickering source and its surround centrally by means of a small black fixation point.

Four sets of flicker determinations were made following 10 min preliminary dark-adaptation. Each set of measurements was, in its turn, begun with $2\frac{1}{2}$ min light adaptation of the eye to the surrounding field and the flickering light, the latter being set above the fusion point. Of the four, the first and last sets of mesurements were obtained with the bright surround, and the second and the third ones with the dim surround. This order was chosen to ensure that practice or any similar effect occurring in the course of the experimental session that might otherwise affect the expected differential c.f.f. under the two surrounds could be eliminated statistically. The intervals between the first two and the last two sets of mesurements were both 2 min, and that between the second and the third ones was 5 min. The subject was dark adapted during each of the intervals. Each set of measurements was made up of 6 flicker determinations based on the method

* It is reasonable to assume that lateral inhibition plays a part in the discrimination of brightness differences between two adjacent visual fields. If so, Trent's (1947) observation that a barbiturate enhanced sensitivity of such a discrimination may lend support to the hypothesis just described.

of limits. Of these, 3 were descending and the other 3 ascending series, running in alternation. In the descending series, the flickering light was first presented well above the fusion point and then lowered slowly until the subject reported the start of flicker; in the ascending series it was altered in the opposite direction, starting from very obvious flicker and ending when the subject saw fusion. In each case, the frequency was varied at a constant rate of approximately 1/2 c/s. in every second throughout the experiment.

Both during light adaptation and flicker observation, the subject viewed the field monocularly through a 2 mm diameter artificial pupil placed immediately in front of the subject's eye. This was done to eliminate possible variation due to differences in pupil size. With each subject, the preferred eye was used.

The subject had preliminary practice in making judgments of the flickering source under conditions similar to those of the actual experiment, except for the intake of the drugs, at the beginning of each of the experimental days.

The drugs used were as follows: one of the following drugs was given to the subject in the morning on each day, and the same one was again given to him about one hour after lunch.

(1) Dexedrine (Dexamphetamine sulphate).

(2) Amytal (Sodium amylobarbitone).

(3) Meprobamate (2-methyl-2-n-propyl-1, 3-propaneidiol-dicarbamate).*

(4) Placebo (lactose).

Doses of the respective drugs given in the morning were as follows: (1) 10 mg; (2) 180 mg; (3) 200 mg; and (4) 450 mg. Doses given in the afternoon were half those given in the morning, except in the case of (4), whose dose was identical on both occasions. All the drugs were given each time in two identically appearing capsules and taken orally.

Of eight subjects, four started the experimental session in the morning, approximately 30 min after the intake of the first portion of the day's drug, while the other four started about the same length of time after the intake of the second portion of the drug in the afternoon. These subjects were engaged in different experiments during the morning. The subject came for testing on 4 consecutive days, receiving a different drug each day. To avoid any positional effect, the four drugs were given to each subject in a balanced randomized order.

The subjects were intelligent, normal adults from various walks of life, and all had volunteered for the test. The age ranged between 22 and 49 with a mean of 34·5. None had gross anomalies of vision and all had a visual acuity of 6/9, or better, either uncorrected or corrected by spectacles, as measured by a Snellen chart.

* This drug was used to see whether another similarly acting central depressant drug (Eysenck, 1960) would produce the same effect as amylobarbitone.

(3) *Results*

The results were analysed separately in reference to: (1) the c.f.f. in the descending series; (2) the c.f.f. in the ascending series; and (3) mean of the c.f.f. in the descending and ascending series. This separate approach was taken, for it was felt that the ascending and descending measurements are not exactly comparable in so far as the physiological and psychological conditions involved are concerned. Although the eye was adapted, before the ascending measurements, to the flickering source set above fusion, it may not be considered strictly equal to starting measurements directly from that state as in the descending series – in the latter case, the adaptation is more direct and immediate. Moreover, some subjects expressed difficulty in making precise judgments in the ascending series. Many reported fusion only to reverse the judgment a moment later, asserting that they responded to it too prematurely. In these cases, the measurements were repeated.

The outcome of the analyses of variance for (1), (2) and (3) is given in summary in Table 9.

TABLE 9

Analysis of variance for the mean c.f.f.'s in the descending (1) and ascending (2) series, as well as the ascending and descending series combined together (3)

Source	*D.F.*	*V.R.*(1)	*V.R.*(2)	*V.R.*(3)
Between Drugs	3	14·353***	3·196*	9·238***
Between Surrounds	1	82·629***	53·727***	81·964***
Between People	7	2·027	20·635***	23·789***
Between Days (within Surrounds)	6	2·108*	4·659**	3·269***
Interaction between Surrounds and Drugs	—	—	—	—

*** significant at. 0.1% level; ** at 1%; * at 5%

As can be seen from it, the effect of the drugs was very marked and produced a significant variance ratio in all three analyses, although it is more marked in (1) and (3), especially in (1).

The mean c.f.f. for each drug based on (1), (2) and (3) are given in Table 10.

TABLE 10

The mean c.f.f.'s in (c/s.) for the results shown in Tables 1, 2 *and* 3, *respectively*

	(1)	(2)	(3)
placebo	23·79	24·61	24·20
dexedrine	24·63	24·48	24·55
amytal	22·03	23·54	22·78
meprobamate	23·22	24·17	23·69

The facilitatory effect of dexamphetamine and the depressive effect of amylobarbitone and of meprobamate are clearly seen, although in (2) the position of the placebo and dexamphetamine are reversed.

The differences between the above means were tested by Duncan's (1955) New Multiple Range Test separately for (1), (2) and (3), and the results are shown in Table. 11.

TABLE 11

Outcome of the multiple range test between mean c.f.f.'s shown in Table 4

	(1)	(2)	(3)
Placebo–Meprobamate	N.S.	N.S.	N.S.
Placebo–Dexedrine	*	N.S.	N.S.
Placebo–Amytal	**	*	**
Amytal–Meprobamate	**	N.S.	*
Dexedrine–Meprobamate	**	N.S.	*
Dexedrine–Amytal	**	*	**

** significant at 1%; * significant at 5%; N.S. not significant.

The differences between the placebo and amylobarbitone, and dexamphetamine and amylobarbitone, are all significant. On the other hand, in none of the comparisons are the differences between the placebo and meprobamate significant. Thus it appears that, under the conditions of the present experiment, amylobarbitone is most effective in altering c.f.f. and meprobamate least, dexamphetamine coming in between them.

The effects of the surrounds are also proved to be highly significant, i.e. either the bright surround depresses c.f.f. or the dim surround enhances it (or both). However, the interaction between the effect of the drugs and the effect of the surrounds is not significant. Nevertheless, as shown in Table 12, when the c.f.f. under the bright surround is subtracted from the c.f.f. under the dim surround, this difference is consistently larger under amylobarbitone than under any other drug or the placebo. No systematic tendency was found with any other drug.

TABLE 12

Differences between c.f.f.'s under the dim and the bright surrounds under each drug

	Placebo	*Dexedrine*	*Amytal*	*Meprobamate*
(1)	1·203	1·385	1·448	1·187
(2)	0·589	0·880	1·203	0·990
(3)	2·063	2·226	2·661	2·177

DISCUSSION

As was explained in the introduction, if the depressant drug enhances lateral inhibitory interaction, then it should either raise or lower c.f.f. depending upon the luminance of the surrounding field relative to that of the test field. If the luminance of the surround is equal to or below that of the test field, c.f.f. should be raised. On the other hand, if the luminance of the surround is above that of the test field, c.f.f. should be lowered.

However, there is another possibility, i.e. the same drug may lower c.f.f *regardless* of the surround conditions, as it did in many studies done without any surround (e.g. Schmidtke, 1951). Apparently our results point predominantly to that direction. In other words, the surround did not alter the basic picture of the action of the depressant drug, i.e., one in which c.f.f. is consistently lowered under diverse external and internal conditions (Berg, 1949; Schmidtke, 1951; Landis and Zubin, 1951; Roback, Krasno and Ivy, 1952). Our finding with dexamphetamine is also in agreement with earlier studies using similar drugs: c.f.f. is markedly raised (Simonson, Enzer and Blankstein, 1941; Simonson and Enzer, 1942; Adler, Burkhardt, Ivy and Atkinson, 1950; Roback, Krasno and Ivy, 1952).

Thus, our prediction regarding the specific action of the drugs in altering the lateral interactions between the test and the surrounding fields in the flicker situation has not been borne out, and, instead, there appeared general depression and enhancement effects under these drugs. However, when we took the *difference* between c.f.f. under the bright and dim surrounds under each drug, we found that amylobarbitone produced the largest difference (Table 12). This might well be a manifestation of enhanced lateral interaction in the visual system produced by amylobarbitone which is normally obscured by the far larger general depressive effect of the same drug. What actually has happened under this drug could be *either* (a) the c.f.f. was less depressed under the dim surround,*or* (b) it was more depressed under the bright surround, *or* else (c) both of these acting together. However, whether this is a real effect or a mere artifact remains to be validated by further work.

Thus, there is no clear proof demonstrated in this study that lateral inhibition was in any way affected by the drugs. Consequently, one is obliged to conclude that the mechanism that was responsible for the general depression and facilitation of c.f.f. (which occurred regardless of the surround conditions) was also responsible for the rise and fall of the threshold of the red stimulus in the Bidwell phenomenon (the identity of the mechanism responsible for this still remains to be discovered), and that the lateral inhibition which might be originated by the white surrounding area was in no way altered by the drugs.

GENERAL CONCLUSIONS

Within the limited scope of the present investigation, certain tentative conclusions may be drawn as to the processes and structure which are responsible for the production of the Bidwell phenomenon.

As pointed out before, it is evident that the phenomenon is a result of some form of neural interaction, i.e. it is not directly due to bleaching or

regeneration of any photo-chemical substances. There has been some indication in the present investigation that the site of this neural interaction is within the retina. The changes in the threshold luminance of the red stimulus following light adaptation are most probably due to some kind of neural reorganization of the retina; as such changes are not likely to affect the phenomenon in question unless the structure responsible for it is within the retina itself, it could be assumed that this might be the site. Furthermore, the close resemblance between the time course of the development of retinal inhibition demonstrated by Granit (1947) and Boynton and Triedman (1953) using the e.r.g. method *and* the specific time arrangement of the two flashes required to produce the Bidwell phenomenon, may lend support to the retinal origin of the effect. If the processes underlying the phenomenon can be located in the retina, the effects of drugs on the thresholds of the red stimulus would be through the drug effects on the retina. It would then be assumed that the drugs used in the present investigation entered the eye with the intra-ocular supply of the blood and acted on the retina directly.

On the other hand, it is equally possible that these drugs may have affected the retina *via* the higher centres. This is suggested from some recent electrophysiological evidence (Granit, 1955; Dodt, 1956; Jacobson and Gestrin, 1958, and Kitai, 1960), as well as from earlier anatomical observations that there are efferent fibres in the optic nerve of some animals. Changes induced by the drugs in the state of the central nervous system can thus influence neural activities in the retina through such efferent pathways. In fact, Jacobson and Gestrin, and Kitai have demonstrated that the effects of barbiturates on the e.r.g. of animals vanish when the optic nerves are severed. In view of the evidence just cited and the fact that the drugs used in the present study have generally strong central effects (Goodman and Gilman, 1955) it may be assumed that the observed effects of the drugs in our study are primarily due to their effects on higher centres. If so, the technique used in the present investigation might be used for assessing the states of the central nervous system.

Chapter 3

"VISUAL MASKING" AND THE EFFECTS OF STIMULANT AND DEPRESSANT DRUGS

H. C. HOLLAND*

MASKING EFFECTS

(1) *Introduction*

A number of experiments have shown that differential energy contours** (Bartley, 1941; Ludvigh, 1953) are necessary for the visual perception of objects (Wever, 1927; Douglas, 1947; Cheatham, 1952; Arden, 1954; Toch, 1956; Smith, 1956; Lindsley, 1958a, 1958b). It has also been shown that contour perception requires time in which to become established, or to lead to percepts *qua* percepts. This establishment time has been examined in a number of ways which are basically variations of the masking procedure and called severally perceptual latency, extinction, sensation time and suppression of the primary visual stimulus. It has been found to be a complex function of brightness, size, retinal area and duration of the stimuli presented. Several explanations of the masking effect, and the analogous metacontrast effect, have been advanced and the locus of the interaction producing them has been sited in both peripheral and central areas (Werner, 1940, 1945; Cheatham, 1952; Alpern, 1952, 1953). The possibility of binocular rivalry has also been considered. It is indeed after carefully considering the arguments in favour of binocular rivalry and rejecting them that Werner (1940) opts to support the notion that the inhibition of a test figure by a masking figure "... does not originate at the retinal level but as a result of a perceptual process at a higher level of integration". The notion of higher integration has also been advanced by Cheatham (1952) in his unsuccessful demonstration of shorter perceptual latency periods for angular contours which were predicted by the satiation theory (Kohler, 1944) and by Lindsley (1958) with a similar phenomenon and a notion of "consolidation" time. On the other hand, the tendency has been for workers dealing with brightness masking (i.e. metacontrast) to support peripheral explanations based upon the interaction of neural elements at the retina or within the optic pathways (cf. Alpern, 1952).

Clearly a large number of factors must determine the formation of visual contours including wavelengths, intensity–brightness, duration, retinal

* The writer is indebted to the Wallace Laboratories for the support of this investigation.

** A contour can be said to exist when two adjacent areas can be distinguished from each other. In Ludvigh's (1953) terms the resolution would depend upon "the ratio of the gradient of retinal illumination to absolute retinal illumination."

position and size of stimuli, being the physical determinants, as well as age, attitude, motivation–fatigue, drugs and other sources of individual differences, to say nothing of the numerous pathological conditions to which the visual and neural systems are subject.

In the half dozen or so experiments which constitute the present report, an attempt is made to examine the suppression of the primary visual stimulus (masking) from the bases of just a few of these factors. The first experiments are concerned with the physical determinants of the stimulus situation, the temporal, the spatial, size and the intensity aspects of the masking effect; whereas the second group of studies investigates the effects of central nervous system stimulant and depressant compounds upon its temporal determinant. These latter studies are an attempt to discover the potentiality of the phenomenon in the field of personality description and are predicted from the "drug postulate" (Eysenck, 1957).

Masking (background)

Masking experiments developed from earlier attempts to measure sensation time or perceptual latency. The basic principles of these methods may be stated as follows; two stimuli are presented in the same sensory area*, first a test stimulus of low energy, followed by a second stimulus of greater energy. When the two stimuli are presented at near simultaneity only the larger (masking stimulus) is perceived and the primary (test stimulus) is said to be masked. If, however, the pair of figures are presented at times which permit an increasing interval between the two, a value of threshold of this interval duration emerges which permits both stimuli to be perceived separately. This interval is then taken as the masking value of the second stimulus and, with some reservations, as the perceptual latency of the first stimulus. It is usually referred to as the "limiting masking interval" (Cheatham, 1952).

Earlier studies were primarily concerned with stimulus energies (e.g. brightness), and it was only later that workers became interested in stimulus contour (Wever, 1927; Werner, 1940, 1945; Cheatham, 1952). Experiments on contour have shown, briefly, the effects of the following:

(a) *Contiguity* – Originally it was thought that with structured stimuli, absolute or near absolute contiguity of the test and masking stimuli was essential (Werner, 1935). However, with masking due to light flashes (metacontrast), authors have reported masking effects over spatial separations of up to one degree of visual angle (Alpern, 1952, 1953), and recently Kolers and Rosner (1960) have reported masking with structured stimuli across spatial separations of 0·63° of visual angle.

(b) *Completeness* – For masking to take place, the immediate juxtaposition of the masking and test contours is not necessary. In other words, the masking of a disk by a ring still takes place if the ring is partially incom-

* Usually the two stimuli are presented in the same phenomenal location with the masking figure acting as a frame for the test figure. (See, however, Experiment 24 in Werner (1935).) This is, however, not the case in metacontrast where the two light areas are separate.

plete or unclosed (Werner, 1935). Under these conditions, the masking effect takes place at different time intervals and the probability of optimal masking decreases. When the completeness of the ring is further diminished, a part of the test stimulus emerges, and, at a critical ratio, the masking effect breaks down completely. Relevant also under this heading is the degree of completeness of the test figure. Werner (1935) attempted to classify the structure of his test figures along a dimension of weak/strong and claimed " . . . the stronger the contour of the inner figure is made by its being built into a stronger pattern, the more this contour will resist absorption by its frame figure." Werner strengthened or weakened his figures by building them into more "impressive figurations".

(c) *Repetition* — Several authors have employed a cycling procedure in which the stimuli are presented in the order disk-pause–ring–pause–disk–pause–ring–pause . . . and so on, the interval between the test and masking stimuli (the interstimulus interval or I.S.I.) (Kolers and Rosner, 1960) being shorter than that between the masking and test stimuli of the next cycle (the intercycle interval or I.C.I.) (Kolers and Rosner, 1960). This repetitive mode of presentation has led to some confusion about whether the masking effect is due to the *retroactive* inhibition of the masking stimulus upon the preceding test stimulus or the *pro-active* inhibition of the masking stimulus by the succeeding test figure (paracontrast). How much, if at all, the masking stimulus influences the suppression of a subsequent complete cycle is not known clearly, although the work of Kolers and Rosner indicates that the probability of seeing the disk is insreased by longer intercycle intervals.

(d) *Size* — One of the aims of the series of experiments to be reported in this paper is to examine the effects of the total size of the stimulus array on masking. Little evidence is forthcoming on this aspect. Several authors have asserted that no masking takes place in foveal areas, whereas others have claimed that although it is more difficult to obtain in the fovea, as opposed to peripheral areas, it can be demonstrated (Werner, 1935; Kolers and Rosner, 1960). Kolers and Rosner were unable to test the foveal hypothesis adequately with their method of investigation, as their smallest possible stimulus subtended a visual angle (0·42°) which was in excess of the Fovea Centralis. On the other hand, Werner (1935) presents experimental results of the gradual diminution of the breadth of the ring until it becomes, phenomenally, a line figure, and of its increase until it becomes, phenomenally, a black disk with a small hole in the centre. This manipulation leads to changes in the time relationships for optimal masking which are due, the author believes, to the qualitative changes in the figure. That is to say, when the outer diameter of the ring is several times larger than the inner diameter there is rivalry between a black disk and what appears to be a white disk; the ratio of the inner and outer diameters being so large that the second stimulus no longer appears as a ring at all.

(e) *Colour* – The colour of the stimuli employed in masking experiments does not appear to be of major importance. Masking is reported (Werner, 1935) as being the same whether black stimuli on white grounds or white stimuli on black grounds are used. Chromatic stimuli have also been used to demon-

strate the effect, but it has been shown that both test and masking stimuli must be of the same hue.* No masking takes place when different hues are used (Werner, 1935), but clearly it would be of interest to discover whether masking occurs when the colours were progressively desaturated. Indeed, as noted above, wavelength is probably an important determinant of masking.

(f) *Shape* — The suppression or masking effect of following stimuli occurs in shapes other than those in which rings and disks are employed. Angular masking shapes will suppress angular test figures so long as they have a contour in common (The interested reader is referred to the experiments of Werner (1935, 1940) who has displayed a large series of masking and test figures including lines, squares, and even some abstract designs.) The major differences between the circular and angular designs appear, from Werner's work** to lie in the shorter time intervals over which the suppression of the figure takes place. It is a finding which has been supported by the work of Cheatham (1952) and Wever (1927).

(g) *Presentation* — Monocular or binocular presentation is not essential for masking as was originally demonstrated by Werner (1940). Kolers and Rosner (1960) have recently reported masking effects when the test stimulus has been presented to one eye and the masking stimulus to the contralateral eye ("dichoptic viewing"). This form of viewing throws new light upon the controversy of whether masking is a central or an entirely peripheral phenomenon, and supports Werner's hypothesis that masking is due to a process at a higher level than the retina. That the periphery, however, does play some part in the process is suggested by the somewhat longer intervals required for optimal masking when the dichoptic method is used.

(2) *Apparatus*

The following description and diagrams are taken from a report by S.E.R. Mable and R.L. Bluffield.*** The same electronic "Dodge" type tachistoscope was used throughout the series of experiments described in this report. It differs somewhat from the Oxford prototype designed by Professor Humphery *et al.* (1955), upon which it is modelled. Wider facilities were permitted by the incorporation of an extra timing stage, which allowed repetitive cycling where necessary (In none of the experiments to be described, however, was repetitive cycling ever used). Ranges covered by the apparatus were light stages 300 msec – 1500 msec, dark and recycle stages 300 msec –4·0 sec; continuously variable.

As with the Oxford instrument, two object cards may be brightly illuminated by banks of alternately pulsed fluorescent tubes, and their images superimposed with the aid of a 45° half-silvered mirror. The fluorescent tubes are standard (AEI Mazda 9^{in}, 6 W) except for the short decay phosphor which is available to order. They are specified to have the characteristic

* The present author has also failed to obtain masking when the stimuli were of different colours.

** According to Werner (1935) angles are "points of concentrated energy of contour and, therefore, offer the greatest resistance to absorption" by an outer figure.

*** Technical staff of the Institute of Psychiatry.

spectrum of magnesium tungstate. They are comparable to daylight but deficient in red. The four tubes are arranged to illuminate a 7^{in}. square and provide a field of about 70 milli-lamberts. The tubes have heater elements at each end and are separately fitted. Provisionis made for periodically reversing the cathode end of the tubes to ensure go od life.

A block diagram is seen in Fig. 1, and a circuit diagram in Fig. 2. Reference to these will probably facilitate the following general description. A pulse, either from "manual" or "recycle", divides at the mixer to tip the Eccles–

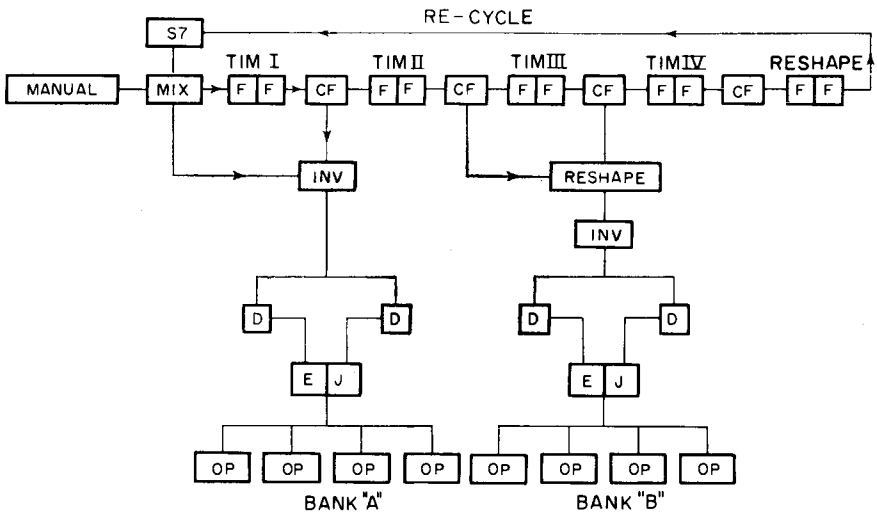


FIG. 1. A block diagram of the timing circuits.

Jordan flip flop on bank "A" as well as triggering timing unit 1. The switching on of the Eccles–Jordan carries the grids of the four ouptut valves positively, causing them to conduct and fire the associated bank of fluorescent tubes. If at a chosen time, timing unit 1 reverts to its stable state, directing a pulse to the Eccles–Jordan, which tips back, cutting off the output valves and extinguishing the light, Then simultaneously, a pulse from timing unit 1 is passed up a cathode-follower to timing unit 2, establishing the interval between bank "A" and bank "B". When timing unit 2 reverts, a pulse dividing at the next cathode-follower goes to trigger timing unit 3 and tip back the Eccles–Jordan on bank "B" so that its fluorescent tubes strike. In due course, timing unit 3 reverts, sending a pulse which extinguishes bank "B" and triggers the recycling of timing unit 4. A pulse from this is reshaped and passed back via switch 7 (S 7) and divided at C 17, C 18 (see circuit diagram) and initiates a further cycle.

Calibration — The short periods of the light and dark intervals were calibrated by the aid of a Cossor double beam oscilloscope and the longer times by a Chronotron electronic clock. Repeated checking showed reliable

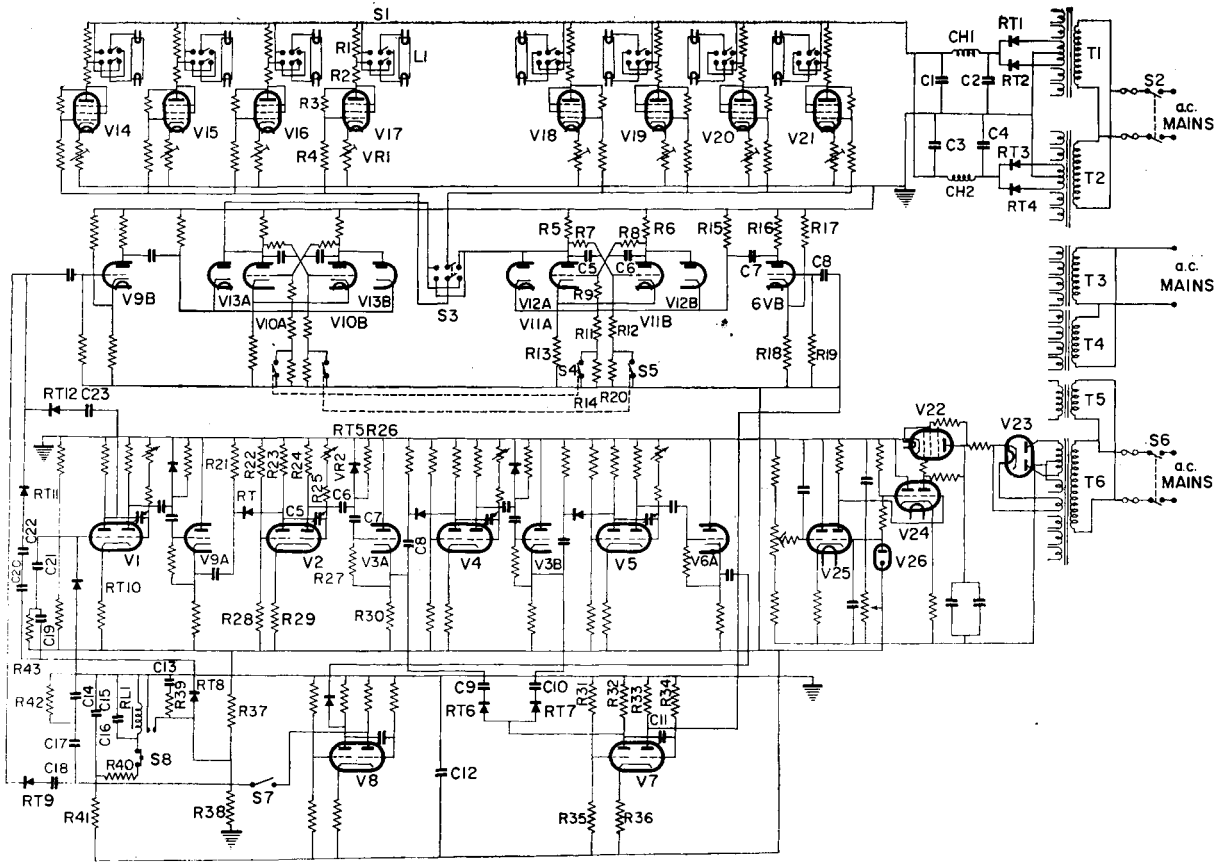


FIG. 2. A circuit diagram. (A detailed list of components may be obtained from the author)

performance, but accuracy greater than that of the "scope method" cannot be claimed.

Originally, the tachistoscope was constructed to carry individual object cards, having slotted carriers behind each bank of light. A further modification, however, was the construction of the display wheels which are seen in Fig. 5. These are large 20″ diameter, duralumin disks faced with matt white PVC sheeting, upon which 4 different sized disks and annuli are mounted. When required, the duralumin wheels are rotated into position and held by a spring–loaded ball bearing and dimple which gives very accurate positioning. It was thus easy to select any of the four disks or the four annuli within a matter of 2 or 3 sec.

SOME PSYCHOPHYSICAL ASPECTS OF MASKING

Experiment 1. Temporal aspects

Introduction – As remarked earlier, it is now well known that sensory processes require time to establish themselves as percepts. If the appropriate time requirements are not allowed between consecutive stimuli, resolution cannot be made and distortions of various kinds take place. This requirement seems to operate at all levels of sensation and perception. Three examples which spring to mind immediately are found in the fusion phenomenon of CFF (Landis, 1954), in the extinction of tactile stimuli (Bender, 1951), and in the suppression of primary coloured stimuli by subsequent achromatic stimulation (Eysenck, 1957). In the first case the distortion takes the form of failure to discriminate discrete stimuli, which fuse and appear in perception as a steady and continuous light. In the second case there is perception of a single stimulus only at one part of the body despite the fact that a second part has likewise been stimulated. In the third case the primary visual stimulus is completely suppressed, although its corollary, a photo-chemical effect, is perceived as a negative after-image.

The distortion or suppression of a primary stimulus is particularly easy to demonstrate in vision where there are contiguous contours for both stimuli (Werner, 1935, 1940).

The investigation to be reported here is an examination of the effect of the duration of presentation of the primary and secondary stimulus upon the interval through which suppression of the primary stimulus or masking occurs. The time intervals numbered six, and were 5, 10, 15, 25, 50 and 70 msec in duration. The dependent variable was thus the duration of the interval in which there was no perception of the primary stimulus.

Subjects – The sample consisted of 83 young males, all between 16 and 18 years. The group was part of a larger population of apprentice engineers employed by a motor corporation. No evidence as to intelligence is offered but, because of keen competition and careful selection, the group was probably slightly above average. Testing was carried out as part of a normal day's work, the Company paying salary and the Institute of Psychiatry paying the costs of transportation and meals. Subjects had no gross defects

of vision and those with corrected vision were permitted to retain their lenses.

Apparatus – The apparatus used has been described more fully above. In this investigation the disk had a diameter of 17 mm (v.a. = 0·913°) and the annulus had inner and outer diameters of 17 mm and 30 mm (v.a. = = 1·161°).

Procedure – Testing was done in almost complete darkness. The subject was seated in front of the instrument and a demonstration given. (The demonstration is important because it is probably at this point that a weakness of the method occurs (see below)). The apparatus was set to give a cycle of 15 msec disk, a 40 msec interval and a 15 msec annulus. The subject was warned to keep his eyes on the fixation light and report what he saw when the instrument was triggered. The setting of 15-40-15 msec which was chosen, because preliminary work had indicated that at this setting all subjects would perceive only the annulus. The setting was then changed to give 15-200-15 msec and all subjects reported the complete sequence of disk–annulus. They were then told that somewhere between the two extreme intervals they would begin to see the disk and that subsequent to each triggering they were to report either "annulus" or "disk–annulus".

It is in this method of reporting that the weakness, referred to above, occurs. Many subjects in the preliminary experiments reported that the disk appeared to "grow" out of the centre until it eventually became the same size as the inner diameter of the annulus, and they required to know when they were to report the disk. Two steps were taken to counteract this source of error: (a) an ascending method of limits was used until the subject reported disk–annulus and then a modified constant method until he reported disk–annulus three times at the same setting; and (b) he was instructed that it was immaterial when he decided to report "a disk" so long as he retained the same criterion throughout the experiment. The constant method was put to the subject as a trick to prevent guessing. After explanation and instruction the subject was given a series of four practice trials before testing began. In the experiment proper, trials were given in the order 10, 15, 25, 50, 70 and 5 msec exposure.

Results – The means and standard deviations of the intervals, during which inhibition or suppression of the disk occurred, for the six trials were as follows: *5 msec*, 71·282, S.D 14·902; *10 msec*, 79·167, S.D. 12·414; *15 msec*, 71·218, S.D. 11·826; *25 msec*, 60·974, S.D. 12·234; *50 msec*, 35·436, S.D. 12·509; *70 msec*, 17·397, S.D. 10·881. These values are presented graphically in Fig. 3.

No test–retest reliability has been assessed for the total group but with a sample of 22 chosen at random and tested at the end of the session on the 10 msec trial, a coefficient of 0·951 was calculated. The retest mean was 1·82 msec lower for this group suggesting a small practice effect. An estimate of the reliability of the present investigation can be made from Table 1, which presents the intercorrelation of scores on the different trials.

It will be seen from Fig. 3 that the 5 msec trial does not maintain the linear function of the remaining five trials and for this reason it has been

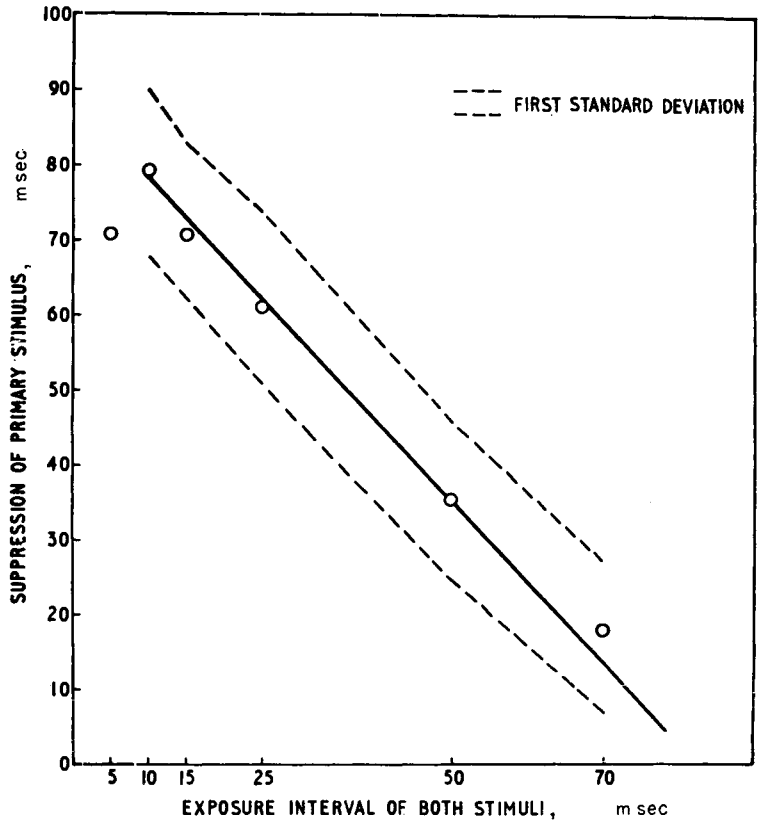


FIG. 3. Graph showing values of interval for suppression of the primary stimulus (masking) for several stimulus exposure periods.

TABLE 1

Intercorrelations of trials scores and derived scores

	1	2	3	4	5	6	7	8
1. Slope		0·147	0·488	0·427	0·202	−0·254	−0·307	0·132
2. 5 msec			0·640	0·651	0·677	0·523	0·563	0·683
3. 10 msec				0·933	0·845	0·635	0·592	0·899
4. 15 msec					0·873	0·674	0·660	0·928
5. 25 msec						0·765	0·703	0·939
6. 50 msec							0·805	0·868
7. 70 msec								0·835

omitted from the calculation of two derived scores which have been designated "Slope" and "Total". The latter score is the sum of effects over the whole investigation. The score "Slope" equals trials $\frac{2+3}{2} - \frac{5+6}{2}$ and represents an index of the decrease in the interval during which suppression will act with increasing disk and annulus durations. The means and standard deviations of the derived scores are: "Total" 264·192, S.D. 53·561; and "Slope" 48·609, S.D. 9·412.

DISCUSSION

By far the most interesting finding of the investigation lies in the almost perfect linearity of the function of inhibition or suppression when plotted against the time exposure of the first and second stimulus. (The first trial must be omitted from this consideration: it is thought that the short period of 5 msec is below the critical duration for the summation of $i \times t$.) An examination of the function suggests that the important feature of the display cycle is not, as might be thought at first, the interval between the end of the disk and the beginning of the annulus but rather the period between the *beginning* of the disk and the *beginning* of the annulus. Taking the mean trial scores as an example it is seen that:

10 msec+79·167 msec=89·167 msec
15 msec+71·218 msec=86·219 msec
25 msec+60·974 msec=85·974 msec
50 msec+35·436 msec=85·436 msec
70 msec+17·397 msec=87·397 msec

Within the errors of assessment this time appears to be nearly a constant. This demonstration would, of course, need to be repeated and investigations conducted to show the influence of the durations of primary and secondary stimuli when they differ in duration.

Experiment 2. Intensity aspects

The next investigation was one in which the effects of the brightness of the stimulus array were examined. The physical stimulus, conditions of size, visual angle, etc., were the same as reported for the earlier investigation, the brightness value being reduced by two neutral density filters with transmission coefficients of log 0·5 and log 1·0. Thus, with a "no-filter" condition, there were three levels of brightness of the total stimulus pattern.

Nine subjects were used, of both sexes, with an age range of 17·50 to 46·75 years. Orders of the three conditions were given in accordance with a 3×3 Latin square, repeated three times. At each level of brightness three trials were taken, at 10, 25 and 50 msec exposure of both primary and secondary stimuli. The order in which the trials were administered was also randomized.

Results – The means and standard deviations of the trials scores under the three conditions for the total group of 9 subjects are presented in Table 2. The values shown in the table are also presented graphically in Figure 4.

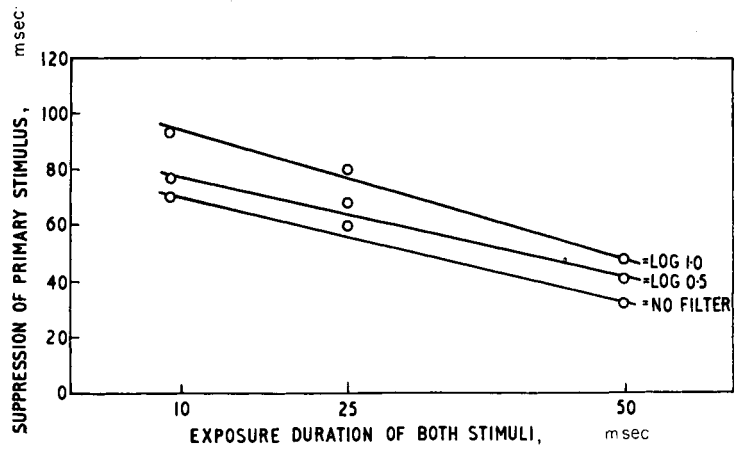


FIG.4. Graph showing changes in the masking interval due to changes in brightness level.

TABLE 2

Means and standard deviations of group scores for the three trials under the three conditions

	A.			*B.*			*C.*		
Exposure	10	25	50	10	25	50	10	25	50
Means									
Upper threshold	74·44	62·78	39·33	85·00	73·89	47·78	101·11	88·33	54·44
Lower threshold	63·33	53·89	27·33	68·89	55·56	32·00	84·44	67·78	35·89
S.D.s									
Upper threshold	14·46	17·34	23·49	22·07	21·62	23·47	20·88	22·08	25·05
Lower threshold	18·03	18·84	19·85	23·42	19·76	17·70	21·71	17·16	18·02

A = No Filter; B = Log 0·5 Filter; C = Log 1·0 Filter.

Several measures were taken and used as scores. These include, as in previous investigations, measures of the *upper threshold* (the point at which two distinct stimuli are perceived), the *lower threshold* (the point at which the two stimuli become a single percept of the annulus), and *the interval of uncertainty* (the threshold between the two levels of clear and certain judgement). This interval may be likened to a hysteresis effect.

Separate analyses of variance have been calculated for these several measures and are outlined in Tables 3, 4, 5 and 6.

TABLE 3

Outline of analysis of variance of upper threshold scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between filters	2	3412·98	61·784	1%
Between people	8	3701·15	67·001	1%
Between order	2	50·24	0·909	—
Between trials	2	11194·09	202·644	1%
Residual	66	55·24		
Total	80			

tab = 4·965, 1%; tac = 11·103, 1%; tbc = 6·137, 1%.

TABLE 4

Outline of analysis of variance of lower threshold scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between filters	2	1520·61	31·945	1%
Between people	8	2922·32	61·393	1%
Between order	2	68·83	1·446	—
Between trials	2	12091·64	254·023	1%
Residual	66	47·60		

tab = 2·113; 5%; tac = 7·739; 1%; tbc = 5·627: 1%;

TABLE 5

Outline of analysis of variance of mean threshold scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between filters	2	2351·87	43·878	1%
Between people	8	3272·44	61·053	1%
Betveen order	2	57·84	1·079	—
Between trials	2	11351·30	211·778	1%
Residual	66	53·60		

tab = 3·518, 1%; tac = 4·261, 1%; tbc = 5·769; 1%.

TABLE 6

Outline of analysis of variance of interval scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between filters	2	464·15	12·208	1%
Between people	8	157·20	4·135	1%
Between order	2	6·78	0·179	–
Between trials	2	11·60	0·305	–
Residual	66	38·02		
Total	80			

tab = 3·662, 1%; tac = 4·726, 1%; tbc = 1·104, 5% NS.

Appended to each outline of analysis of variance is the breakdown of the overall variance attributable to the interposition of the filters. In all but one case, (the difference between the second and third condition for the interval of uncertainty) the individual *t*-tests are clearly significant. The mean scores with the differences between them clearly show that the duration of time over which supression of the primary stimulus occurs is significanty lengthened by a reduction in the brightness of the stimulus pattern.

Bartley (1941) suggests that visual acuity is scarcely a reality until contours form, and, as acuity is also dependent to a large extent upon brightness, it might be suggested that longer masking intervals at lower brightness levels are related by way of acuity.

Experiment 3. Spatio-temporal aspects

The third aspect of masking to be examined might be broadly stated as the spatio-temporal. As outlined earlier, some authors have contended that for adequate masking to take place, the contours of the masking and test stimuli have to be in immediate juxtaposition, whereas others have shown that masking can be accomplished when they are removed from each other (c.f. Kolers and Rosner, 1960). It has been to throw some further light upon this problem that the present study has been conducted.*

Four rings or annuli were constructed from black matt paper and pasted upon the white plastic (non-shine) surface of a circular steel disk which could be rotated within one of the viewing apertures of the tachistoscope. The second viewing aperture contained a similar construction for the test stimuli. The rings had inner diameters of 5/8 (0·625) in. and outer diameters of 2¼ in. (No. 1), 2 in. (No. 2), 1½ in. (No. 3) and 1¼ in. (No. 4) which subtended visual angles of 1·25°, 1·11°, 0·83°, and 0·69°. The test stimuli

* It is known, for instance, from the works of Ratcliff, Miller and Hartline (1958) that inhibitory effects (among receptor units) are less in far widely separated areas than in areas closer together.

which took the form of solid black circles on a white ground, had diameters of 5/8 in, 9/16 in, 1/2 in. and 7/16 in. and subtended visual angles of 0·35°, 0·31°, 0·28° and 0·24°. Thus, for any of the four differently sized rings, the test stimulus could be presented either contiguously with the masking stimulus or removed at three levels representing contour differences of 1/32 in, 1/16 in or 3/32 in.

Eight subjects were employed in the experiment, all being young males between the ages of 15 and 17 years. Subjects were administered the experimental regime in accordance with a 4×4 Latin square, repeated twice. There were four test stimuli for each ring size and these were assessed for the masking interval by the same randomization procedure. That is to say, both the rings and the test stimuli were presented in the order demanded by the Latin square. The possibility of fatigue prohibited a testing session of too great a length of time and only single ascending thresholds of the critical masking interval were taken. Even with individual presentations of the paired stimuli every 5 or 6 sec and a rest period of 3 min between annuli, the present length of testing, about 40 min, was quite long enough for the subjects to maintain attention on the display.

Results – The means for the masking interval determined by the four differently sized annuli were as follows:

Ring 1	*Ring 2*	*Ring 3*	*Ring 4*
81·56 msec	79·69 msec	77·50 msec	78·75 msec

As will be seen there is a tendency for the larger rings to produce longer masking intervals, the apparent reversal of this tendency being due entirely to one subject.

The mean masking intervals for the four test stimuli were:

Disc 1	*Disc 2*	*Disc 3*	*Disc 4*
85·31 msec	81·41 msec	77·03 msec	73·75 msec

Once again there is a clear reduction in the critical masking interval due to the progressive removal of the disk from absolute contiguity with the ring.

The overall differences in scores due to variations in the independent variable of the design have been examined, once again, by analysis of variance. The outline of the analysis is presented in Table 7. An examination of this table indicates that several sources of variance have been extracted: *Subjects*, or individual differences; *Orders*, or practice effects; *Rings*, variance due to differences in annulus size; *Disks*, variance due to differences in the size of test figures and their removal from contiguity with the masking stimulus; and finally, the interaction between *Disks* and *Rings*. With the exception of the interaction, all sources of variance are significant. The interpretation of most of them is quite clear from the design. The order effect indicates a practice effect which is also supported by the means

TABLE 7

Outline of analysis of variance of the critical masking interval scores when annuli are varied at four levels and test stimuli at four levels

Source	*D.F.*	*S.S.*	*M.S.V.*	*F.*	*P.*
Between subjects	7	12431·00	1775·86	57·66	1%
Between orders	3	427·00	142·33	4·62	1%
Between rings	3	281·00	93·67	3·04	5%
Between discs	3	2459·00	819·67	26·61	1%
Interaction discs x rings	9	160·00	17·78	N.S.	
Residual	102	3142·00	30·80		
Total	127	18900.00			

of the four orders, i.e. 82·03, 79·21, 79·37 and 76·87 msec. There is a very large subject effect, denoting individual differences which range from 238·75 msec to 383·75 msec for the 16 trials. The two main independent variables, the ring and disk sizes both produce significant F ratios when tested against the general residual. It may be argued that the more appropriate "error", against which to test these main effects, is the ring–disk interaction. If this is done, however, the F ratio in fact increases.

The observed results are consistent with earlier work which has shown masking effects with disparate stimuli both in vision and somesthesis (Uttal, 1960).

Experiment 4. Size aspects

The final aspect of masking investigated during the preliminary stages was the change in the critical masking interval due to increases in the size of the masking ring. This investigation had been suggested by a source of variance, significant at the 5% level, outlined in Experiment 3.

In this study the test stimulus was held constant with an outside diameter of 5/8 in (va = 0·35°) and the masking stimuli changed at four levels of inner diameter 5/8 in and outer diameters of 1¼ in, 1½ in, 2 in and 2¼ in. It was thus identical with one of the trials of the preceding experiment but was investigated more systematically.

There were 8 subjects in the experiment. Once again, they were part of the apprentice population which has been used throughout, although, of course, they had not been tested previously. The ages of the group ranged between 15 and 17 years, and none of them had corrected vision.

The procedure, in which only ascending trials were used, was the same as has been outlined above. Three upper threshold measures were taken at

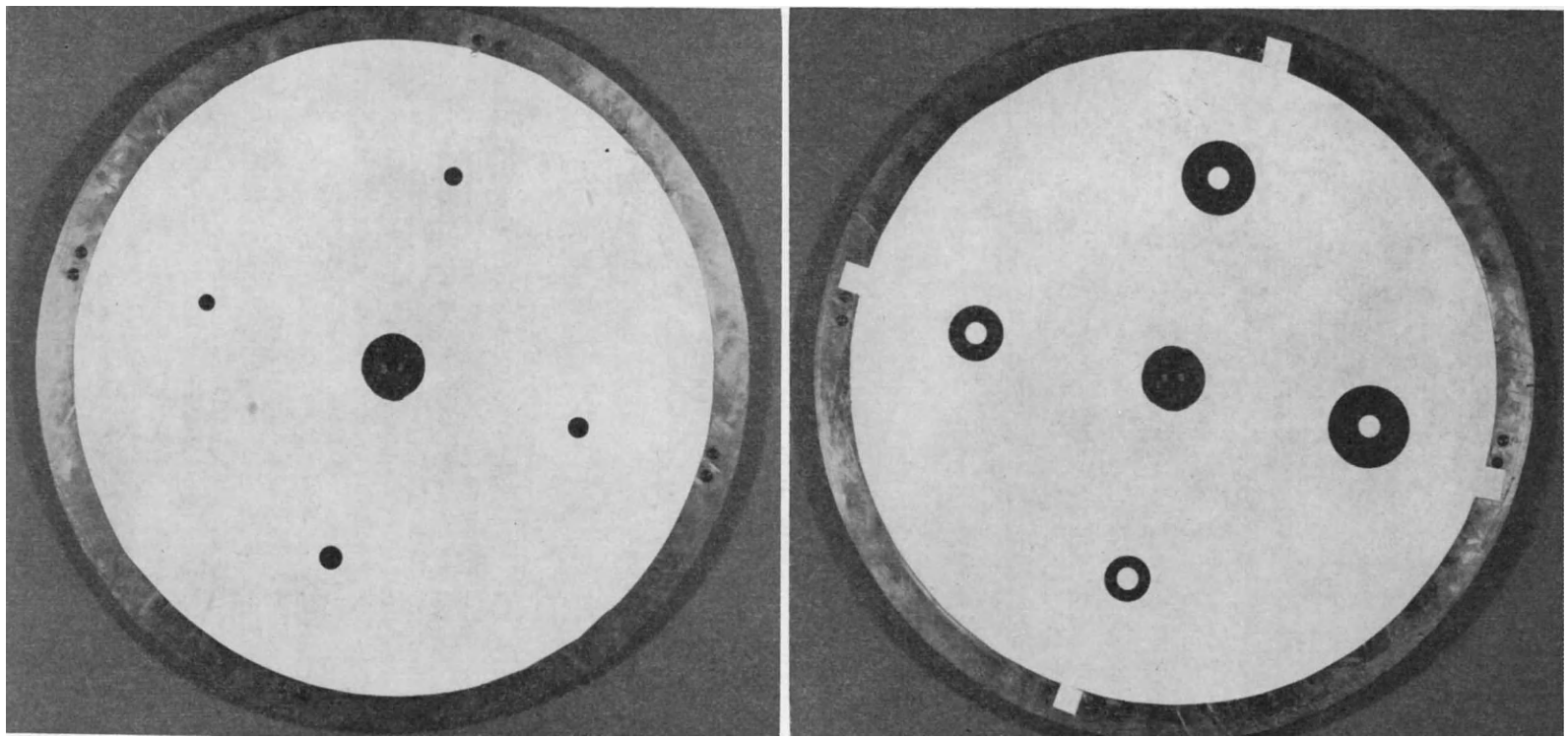


FIG. 5. Photograph of front surfaces of both disks.

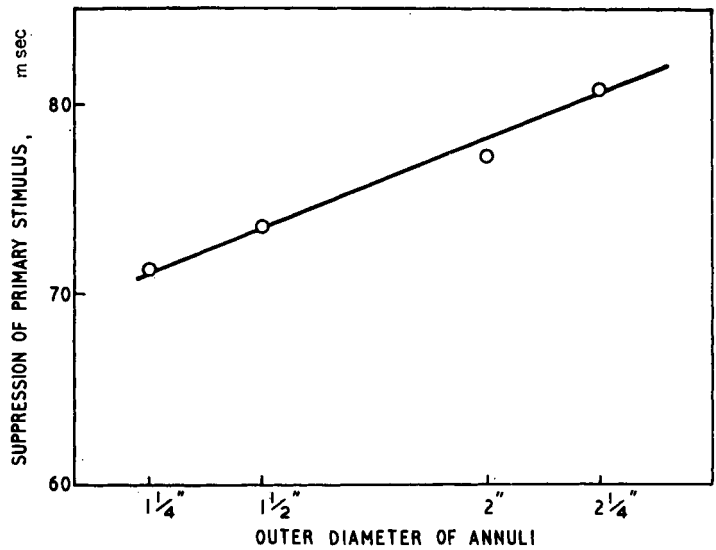


FIG. 6. Changes in the masking interval due to increases in the size of the masking stimulus.

each of the four masking ring sizes, the order of presentation being randomized in accordance with a 4×4 Latin square, administered twice. The apparatus has already been described.

Results — The mean masking intervals of the four rings for the 8 subjects are as follows:

Ring No. 4 (1¼ in).	*Ring No. 3 (1½ in).*	*Ring No. 2 (2 in).*	*Ring No. 1 (2¼ in).*
71·46 msec	72·70 msec	77·08 msec	81·25 msec

The intervals show a clear increase in value as the size of the masking ring increases. The points lying along a straight line when compensation along the abscissa, for the ½ in jump between Nos. 3 and 2 is made (see Fig. 6).

The individual variation between scores has again been assessed by analysis of variance, and the outline is presented in Table 8. In the analysis, the variance due to individual differences (Between "*People*") is again highly significant, there is no "*Order*" effect despite the fact that the mean scores tend to decrease very slightly with successive trials, and the main independent variable (Between "*Rings*") is very highly significant. Using a variance ratio test the individual comparisons are presented as *t*-tests in the body of the table. They show that all means are differentiated from each other with the exception of the comparison between Rings 4 and 3; i.e. the comparison between 1¼ in and 1½ in outer diameters.

The results of the experiment are found to support more strongly the indication outlined in Experiment 3.

TABLE 8

Outline of analysis of variance of the critical masking interval for four sizes of masking stimulus

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between people	7	1123·20	19·14	1%
Between orders	3	41·67	·71	N.S.
Between rings	3	1431·25	24·39	1%
Residual	18	58·68		
Total	31			

Sizes: $t_{43} = 0{\cdot}979$, N.S; $t_{42} = 4{\cdot}405$, 1%; $t_{41} = 7{\cdot}668$, 1%; $t_{32} = 3{\cdot}426$, 1%; $t_{31} = 6{\cdot}689$, 1%; $t_{21} = 3{\cdot}264$, 1%;

GENERAL SUMMARY OF EXPERIMENTS 1 – 4

Temporal aspects — It is clear from the results of varying the duration of exposure that the masking phenomenon is highly reliable. As stressed previously, the important fact to emerge is the constant period calculated from the beginning of the first (test) stimulus to the beginning of the second (masking) stimulus. This function breaks down at stimulus exposures below 10 msec and the explanation suggested lies in the period in which there is the summation of $i \times t$. Indeed, even at the purely subjective level, the 5 msec pulse appeared less bright than the second shortest duration (10 msec). That such a simple explanation, i. e. reduction in intensity, is not completeiy adequate however, is suggested by the second experiment in which the interposition of neutral density filters led to an increase in the duration of masking.

Intensity — The analyses of the results of intensity reduction by filters indicates a very clear lengthening of the masking interval as the intensity is reduced. The explanation of this brightness effect is not as simple as was first thought. Indeed, the notion advanced for the failure of the 5 msec exposure to maintain the linearity of the masking function delineated by longer exposures, namely, a Bunsen–Roscoe effect due to the incomplete summation of $i \times t$, is contradicted by the observed results of intensity reduction. Initially, therefore, one is forced to accept some "common sense" hypothesis which would account for the longer masking intervals with reduced brightness, in terms of reduced visual acuity and a larger discrimination threshold at the lower brightness levels. Reduced brightness of the stimuli should lead to poorer contours and their easier elimination or appropriation.

Spatio-temporal — As remarked earlier, the progressive removal of the test stimulus from the masking stimulus was designated the "spatio-temporal" aspect because to mask, or inhibit, the disk the inhibitory effects of the annulus had to irradiate across an area of non-stimulation.

The results indicate fairly clearly that masking does take place across such areas and the reduction in the masking interval appears to be proportional to the magnitude of the "gap".

The results are also in line with the findings of Uttal (1960) on the effects of spatial removal in somesthetic masking.

Size effects — The analysis of variance carried out on the results of testing the spatial aspects of masking had indicated the influence ofincreasing the physical size of the masking stimulus. This function was further investigated in Experiment 4. As the results indicate, the masking interval increases as the size of the masking figure is increased and five of the six possible comparisons between the four figures are highly significant. Presented graphically (Fig. 6), the four points suggest a fairly clear straight line function which could, and should, be extended in the light of Werner's finding that there are qualitative changes occurring as the ring is made larger.

THE EFFECTS OF CERTAIN STIMULANT AND DEPRESSANT DRUGS UPON THE TEMPORAL COURSE OF VISUAL MASKING

Experiment 5. The effects of sodium amylobarbitone and dexamphetamine sulphate on masking of a visual stimulus.

A theory which will give some account of individual differences in an inhibitory function is found in Eysenck's theory of extraversion–introversion (Eysenck, 1957). This theory asserts that differences in molar patterns of behaviour are determined by an excitation–inhibition balance of the central mechanisms mediating S-R connections: the extravert being characterized by high inhibitory potentials and low excitation and the introvert being characterized by low inhibitory potentials and high excitation. A corollary of the extraversion theory is found in the drug postulate (Eysenck, 1957, 1960) which asserts that depressant drugs (sedative and hypnotic) will increase inhibitory potentials, reduce excitation and thus have an extraverting effect. Conversely the stimulant drugs (amphetamine, caffein, etc.) will reduce inhibition and raise excitation and thus have an introverting effect. It is from the extraversion theory and its derivative drug postulate that the outcome of the present investigation is predictable and may be stated as follows:

If the duration of the time over which suppression of the primary stimulus occurs is a function of the inhibitory influence of the secondary stimulus; and, if the magnitude of developed inhibitory potentials may be changed by depressant and stimulant drugs; then sodium amylobarbitone, a depressant drug, will increase, and dexamphetamine sulphate, a stimulant drug, will decrease the duration of the interval in which suppression of a primary stimulus occurs.

Subjects — To test the hypothesis outlined 6 subjects were used. Each was tested on three occasions with at least 24 hours between testing sessions. The treatments, comprising the two drugs and a placebo control, were

administered in accordance with a Latin square design which counteracted any differential learning effects. The subjects were all female, and their ages ranged from 19 to 24. Testing began not less than 1½ and not more than 3½ hours after the ingestion of the drug capsule. Each subject was examined daily by a physician both before and after the testing programme. Volunteers were excluded who displayed any obvious physical defect, had hay fever or any allergy.

The drugs — The drugs were as follows: sodium amylobarbitone, 195 mg; dexamphetamine sulphate 10 mg; placebo.

All capsules were identical in appearance and were taken orally. At least 1½ hours was allowed for absorption before the individual was subjected to any experimental procedure.

Procedure — Six trials were taken by each subject on each of the 3 days. A single threshold was determined at 5, 10, 15, 25, 50 and 70 msec; these being the duration periods of both disk and annulus. The upper threshold was taken as the length of the interval between disk and annulus presentation at which the disk appeared as a percept.

Results — The results for the six trials under the three treatment conditions are presented in Table 9. For clarity the mean trial scores are presented graphically in Figure 7.

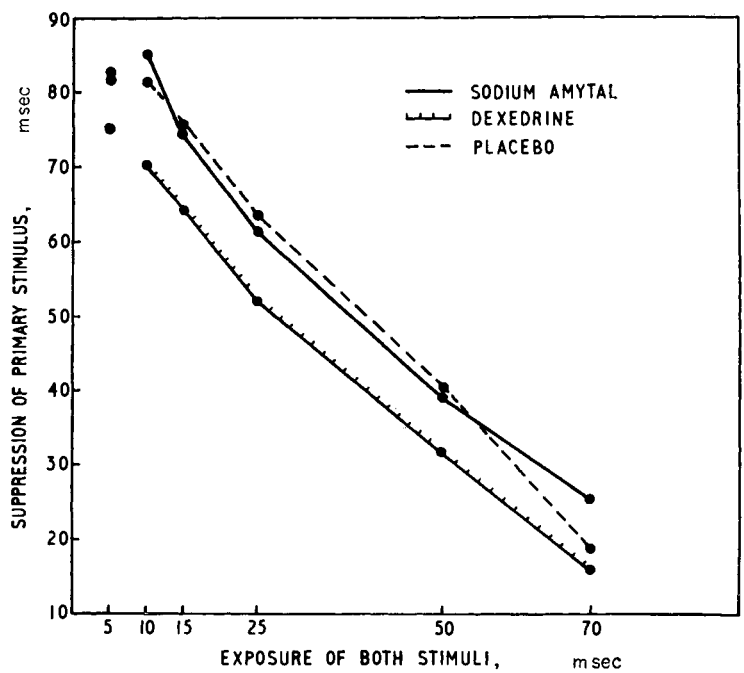


FIG 7. Graph showing effect of stimulant, depressant and placebo treatment on the masking interval.

TABLE 9

Subjects	*Order*	Placebo						Dexedrine						Amytal					
		5 msec	10 msec	15 msec	25 msec	50 msec	70 msec	5 msec	10 msec	15 msec	25 msec	50 msec	70 msec	5 msec	10 msec	15 msec	25 msec	50 msec	70 msec
1	1 2 3	55	75	65	50	22	1	50	55	50	30	12	1	50	65	50	30	4	1
2	2 1 3	120	105	100	85	65	25	90	80	75	65	50	30	100	105	95	80	55	40
3	1 3 2	85	80	75	70	45	25	50	50	50	45	15	1	90	80	75	70	45	28
4	3 1 2	75	70	65	55	30	17	85	65	60	55	27	14	80	80	65	55	26	15
5	2 3 1	95	95	90	80	60	40	105	105	95	85	70	50	95	105	95	80	70	50
6	3 2 1	65	70	55	40	22	5	70	65	55	35	20	4	80	75	65	55	35	20
MEAN		82·5	82·5	75·0	63·3	40·7	18·8	75·0	70·0	64·2	52·5	32·3	16·7	82·5	85·0	74·2	61·7	39·2	25·7

The scores presented have also been subjected to an analysis of variance; the outline of which is presented in Table 10. All the sources of variance are

TABLE 10

Outline of analysis of variance

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between people	5	5351·6372	95·88	1%
Between drugs	2	1009·3426	18·08	1%
Between trials	5	10600·5704	189·92	1%
Between orders	2	655·7315	11·75	1%
Residual	93	55·8172		

t Placebo x Dexedrine = 4·9384 p = 1%; t Placebo x Amytal = 0·5049 p N.S.; t Dexedrine x Amytal = 5·4433 p = 1%

highly significant. The variance between "*People*" is, of course, a quantification of the considerable individual differences between subjects which is known to exist for the masking phenomenon. The "*Trials*" variance represents the change in the duration of the interval over which suppression of the primary stimulus occurs with increasing periods of presentation of the two stimuli. The "*Order*" effect must be considered as a practice effect. When the trials were summed for each subject and examined for change from day 1 to day 3, the scores were: *day 1*=2199 msec; *day 2*=2142 msec; and *day 3*=1909 msec, showing a gradual but consistent decrease over the time in which inhibition occurred with increased practice. The "*Drug*" effect variance represents the main experimental hypothesis. The overall variance for treatments can be broken down into three comparisons. Using a variance ratio test, the *t* values of the comparisons are presented in Table 10 with the outline of the analysis of variance. They indicate that although there are highly significant differences between Dexedrine and Amytal, and between Dexedrine and Placebo, there is no acceptable difference between Amytal and Placebo.

A derived score, "*Slope*", was extracted from the trial scores for each subject. Defined as trials $\frac{2+3}{2} - \frac{5+6}{2}$, it was intended to express the slope of the decreasing function of the period of suppression of the primary stimulus due to the increase in the duration of stimulus presentation. The values of "*Slope*" are also presented in Table 11. Analysis of variance of "*Slope*" scores (Table 12) failed to reveal any significant drug effect.

As in an earlier study, the 5 msec trial failed to display a value which maintained the same linear function as the remaining trials. There is here an exception in the case of Dexedrine. The explanation previously advanced was that 5 msec is below the critical duration for $i \times t$. Because the function maintains itself for the Dexedrine treatment, the finding is suggestive of a shortening of $i \times t$ under the stimulant drug.

Conclusions

The original prediction that the depressant drug would lengthen and the stimulant drug shorten the time interval during which the suppression of the primary stimulus will occur has been fulfilled in most of its particulars. The difference between the results of testing between Dexedrine and Amytal treatments is highly significant, as is the difference between Dexedrine and Placebo. The one particular in which the prediction is not fulfilled is that the expected difference between Amytal and Placebo does not achieve a statistical level of significance. The "Placebo effect" is a

TABLE 11

Table of "Slope" scores under the three conditions

	Placebo	*Dexedrine*	*Amytal*
1.	58.5	45.0	55.0
2.	57.5	37.5	52.0
3.	42.5	42.0	52.0
4.	44.0	42.0	52.0
5.	41.5	40.0	40.0
6.	49.0	48.0	42.5
Total	293.0	254.5	283.0
mean	48.83	42.42	47.17

TABLE 12

Outline of analysis of variance of "Slope" scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between people	5	62·68	2·315	N.S.
Between drugs	2	66·51	2·456	N.S.
Between orders	2	24·18	–	N.S.
Residual	8	27·077		

finding which may support Werner's (1935) contention that attitudinal factors are important in this kind of perceptual phenomenon.

The differences due to the treatments and the lack of difference between the derived score "slope" on the three occasions suggest that the decreasing

function of the period of suppression of the primary stimulus, (by increasing periods of stimulus duration), is pushed along its axes *in toto* without changing its angles of intercept. Indeed, such similarity of change on all the trials is reminiscent of an intensity effect of the kind observed in flicker fusion.

The result of the experiment must also be considered as supporting the derivative postulate of the Extraversion theory although the failure of the depressant drug to suppress the primary stimulus over a longer period than the placebo still requires explanation.

Experiment 6. The effects of meprobamate on masking

This was an investigation in which the effects of the ataractic meprobamate (2-Methyl-2-n-propyl-1; 3 propaneidiol dicarbamate) were examined in relation to suppression, or otherwise, of the primary visual stimulus (i.e. masking). The choice of the drug was due to the fact that in other perceptual tasks, (e.g. apparent movement after-effects, critical flicker fusion, etc.,) experiments by the present writer (Holland, 1959) had shown meprobamate to be a non-specific depressant which differs from the better known depressant barbiturates (e.g. Amytal) quantitatively rather than qualitatively (Eysenck, 1960), and it was predicted that if the phenomenon under consideration was of an inhibitory nature, its parameters would be altered by the central depressant action of the drug.

The drug — What information is available concerning meprobamate suggests that it must be classed with other ataractics derived from either phenothiazine, the Rauwolfia alkaloids, benzhydrol or mephenesin, (meprobamate being a mephenesin derivative). The compound is said to be a non-toxic, non-addicting, non-depressant relaxant, (Margolis, 1957; Litchfield, 1957; Haizlip, 1958), differing from the barbiturates in its site of action, its side effects, and its tranquillization without confusion. The drug has been used in several abnormal, tension and emotional states (Marquis 1957; Dickel, 1957; Litchfield, 1957).

Subjects — The subjects in this study numbered 10. There were 5 males and 5 females, their ages ranging from 17·00 to 32·45 years. Subjects were tested on each of 2 days, receiving either 800 and 200 mg of meprobamate or an equal amount of placebo. The second and smaller dose was given between $3^1/_2$ and 4 hours after the administration of the larger dose and the present test approximately 30—45 min after the second dose of 200 mg.

Conditions of testing were as in earlier experiments except that six trials were administered in the order 10, 15, 25, 50, 5, 70 msec. exposure.

Results — The results of scoring the upper and lower thresholds are presented with their standard deviations in Table 13. They are presented graphically in Fig. 8.

TABLE 13

Meprobamate — Placebo
Means and standard deviations of scores for the six trials for the ten subjects under the two treatment conditions

	Placebo						*Meprobamate*					
Trials	5	10	15	25	50	70	5	10	15	25	50	70
Upper threshold	75·0	72·5	67·0	59·5	35·5	18·0	87·5	88·0	80·0	70·0	47·9	28·6
S.D.	17·8	13·2	12·7	11·7	15·2	14·5	26·5	17·2	16·7	16·2	19·2	17·7
Lower threshold	62·5	62·0	56·0	51·0	25·1	9·9	76·5	73·0	68·0	57·5	37·8	18·9
S.D.	17·7	11·4	13·5	13·9	16·0	13·6	25·7	18·6	14·2	13·8	17·1	16·3

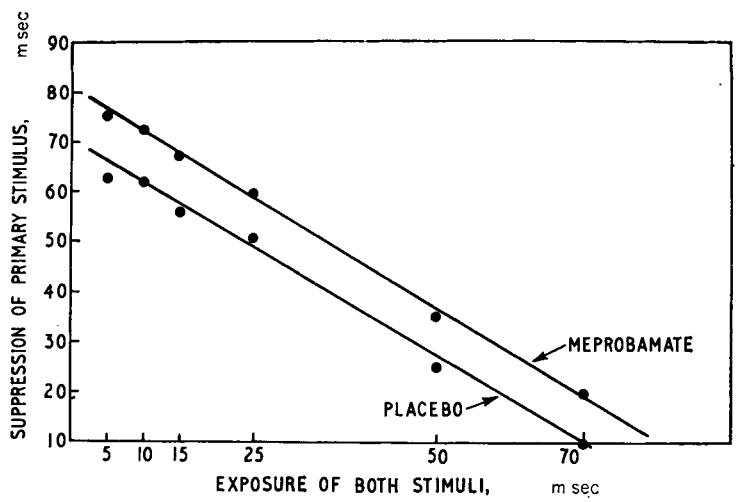


FIG. 8. Differences in the masking interval under placebo and Meprobamate conditions.

Individual simple analyses of variance have been calculated for the scores taken or derived, as in the other investigations, and their outlines presented in Tables 14, 15, 16 and 17. With one exception, namely the interval of uncertainty scores, a similar pattern emerges from all the analyses: (a), the variances for *people* are very significant; and (b), differences due to the *Drug* effect are significant, albeit at a somewhat lower level. In other words, meprobamate has produced effects on masking which are essentially the same as the better known and more profound depressant Amytal. In the one particular in which the expected results failed, namely the failure of the differences in the interval of uncer tainty scores due to the drugs, to reach significance there is a tendency for the placebo thresholds to be better discriminated than those of the drug.

TABLE 14

Meprobamate — Placebo
Outline of analysis of variance of lower threshold scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between drugs	1	3542·53	6·558	5%
Between people	9	2217·76	4·106	1%
Between order	1	1·63	0·003	–
Residual	108	540·16	–	
Total	119			

TABLE 15

Meprobamate — Placebo
Outline of analisys of variance of upper threshold scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between drugs	1	4625·21	7·568	5%
Between people	9	2225·25	3·641	1%
Between order	1	12·68	0·021	–
Residual	108	611·19		
Total	119			

TABLE 16

Meprobamate — Placebo
Outline of analysis of variance of mean threshold scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between drugs	1	4065·85	7·268	5%
Between people	9	2353·71	4·207	1%
Between order	1	5·85	0·011	–
Residual	108	559·45		
Total	119			

TABLE 17

Meprobamate — Placebo
Outline of analysis of variance of interval of uncertainty scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between drugs	1	72·08	3·172	–
Between people	9	66·33	2·919	5%
Between order	1	5·21	0·229	–
Residual	108	22·73		
Total	119			

Experiment 7 (a) and (b). The effects of nitrous oxide and oxygen on masking

The third study of the effect of drugs on the suppression of a visual stimulus is one in which fixed proportions of N_2O, oxygen, and an air placebo were administered to a small group of subjects. Despite the limited use of nitrous oxide in psychological research (Steinberg, 1955, 1956), this anaesthetic drug was chosen for the investigation for several reasons: (a) with suitable instrumentation it is easy to administer and easy to control to a considerable degree of accuracy; (b) it acts rapidly to achieve its maximum effect within seconds; (c) recovery from the drug is rapid and, within certain limitations,there are no after, or side, effects; (d) as a corollary of the rapid recovery, different treatment conditions may be undergone by subjects within a time short enough to prevent gross changes of their physiological and psychological environments; and (e) acting as it does to depress the central nervous system,* the drug is similar to those which have supplied evidence in verification of the drug postulate.

The present investigation is composed of two related experiments on the same group of 6 subjects. The description and results will be presented separately.

Experiment (a)

In the first experiment 6 subjects (3 males, 3 females) whose ages ranged from 20 to 31 years were administered, at one session, the following treatments:

(a) 30 per cent N_2O (in oxygen);
(b) 100 per cent Oxygen;
(c) Air;

* The reader is referred to Chapter 15 for an elaboration of this statement.

TABLE 18A

First Square

Sub.		N_2O		Oxy		Placebo
1	123	AIR 5 min Oxy 5 min N_2O 10 min Test	3 min Oxy 4 min Air	3 min Oxy 10 min Test	5 min Air	10 min Test
2	312	Placebo		N_2O		Oxy
		AIR 3 min Air 10 min Test	5 min Oxy	5 min N^2O 10 min Test	3 min Oxy 4 min Air	3 min Oxy 10 min Test
3	231	Oxy		Placebo		N_2O
		AIR 3 min Oxy 10 min Test	10 min Air	10 min Test	5 min Oxy	5 min N_2O 5 min Oxy 10 min Test

1 = N_2O; 2 = oxygen; 3 = Air (Placebo).

TABLE 18B

Second Square

Sub.		Oxy		N_2O		Placebo
4	213	AIR 3 min Oxy 10 min Test	5 min Oxy	5 min N_2O 10 min Test	3 min Oxy 4 min Air	10 min Test
		N_2O		Placebo		Oxy
5	132	AIR 5 min Oxy 5 min N_2O 10 min Test	3 min Oxy 4 min Air	10 min Test	5 min Oxy	3 min Oxy 10 min Test
		Placebo		Oxy		N_2O
6	321	AIR 10 min Test	5 min Oxy	3 min Oxy 10 min Test	5 min Oxy	5 min N_2O 5 min Oxy 10 min Test

all of which were flavoured with oil of lavender. The regime was as shown in Table 18. The dependent variable was the duration of the interval over which suppression of the primary stimulus occurred at three levels of exposure of both primary (test) and secondary stimuli, i.e. 10, 15, 50 msec.

Apparatus – The apparatus employed for administration of the required conditions was a commercially available Boyles Table (British Oxygen Company) modified to deliver compressed air instead of cyclopropane. Delivery of all gases was through a R.A.F. oxygen mask containing a microphone. The subjects' responses of "disk", "annulus", or "disk-annulus", plus any relevant speech were taken from the mask microphone and amplified, making them audible to the experimenter.

Experiment (b)

A second measure of the effect of nitrous oxide was taken in the afternoon following experiment (a). In this case, a series of measures of the duration of the masking effect were taken during a period of approximately 30 min. The values of the measurements were assessed as quickly as was consistent with the method of discrete presentation, but varied between about 3/4and $1^1/_2$min. The duration of both disk and annulus presentations was fixed at 10 msec. During the period of 30 min, 4 treatment conditions were administered in the order; air, oxygen (100 per cent), N_2O (20 per cent) and air. The original air inhalation was designated "air_1" and the final inhalation "air_2".

Times were noted at the end of each threshold determination and also at each point when a gas switch over (i.e. air to oxygen or oxygen to N_2O) occurred. At the end of the experiment, the scores were plotted to give a complex curve of the effect of the four treatments. From it were abstracted a set of scores which represented either the exact or interpolated scores at 1, 2, 3 and 4 min for air_1; 1, 2, 3 and 4 min for oxygen; 1, 2, 3, 4, 5, 6, 7 and 8 min for N_2O; and 1, 2, 3 and 4 min for air_2; after the onset of the respective treatments.

Results – The means of the ascending and descending thresholds for the three durations of stimuli in the first part for the drug conditions were as follows: (see also Fig. 9.)

		Oxygen	*Air*	*N_2O*
Threshold	msec	53·028	64·472	90·278
Upper threshold	"	57·883	71·556	110·556
Lower threshold	"	47·667	57·389	70·000
Interval	"	9·611	14·167	40·556

Changes from treatment to treatment have been assessed by analysis of variance for each measure taken or derived from the scores and are presented in Tables 19, 20, 21 and 22.

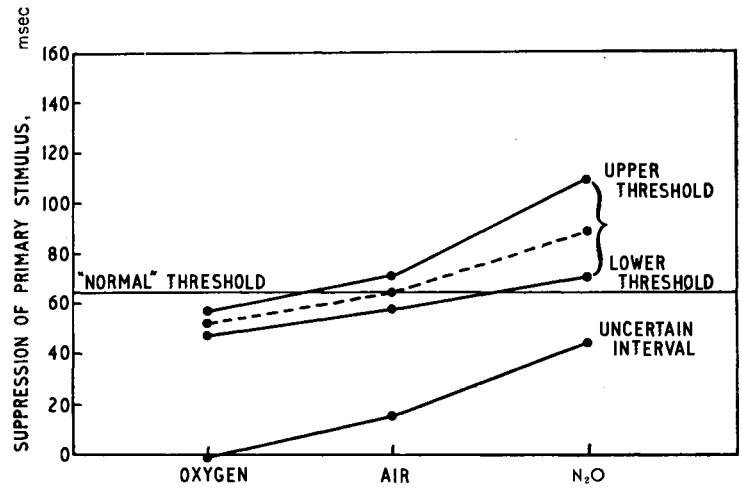


FIG. 9. The effects of air (Placebo), Oxygen and Nitrous Oxide treatment showing changes in the masking interval and impairment of discrimination.

TABLE 19

Outline of analysis of variance of threshold scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between drugs	2	6553·40	51·30	1%
Between people	5	5338·95	41·79	1%
Between trials	2	6213·31	48·64	1%
Between orders	2	606·06	4·74	5%
Residual	42	127·75		
Total	53			

t, N_2O/oxy. = 9·889; 1%

t, N_2O/air = 6·851; 1%

t, oxy/air = 3·038; 1%

TABLE 20

Outline of analysis of variance of interval of uncertainty scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between drugs	2	5024·06	13·35	1%
Between people	5	1428·80	3·80	1%
Between trials	2	369·06	0·98	N.S.
Between orders	2	452·06	1·20	N.S.
Residual	42	376·26		
Total	53			

t, N_2O/oxy. = 4·787; 1%
t, N_2O/air = 4·082; 1%
t, oxy/air N.S.

TABLE 21

Outline of analysis of variance of lower threshold scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between drugs	2	2257·02	23·28	1%
Between people	5	3714·28	38·30	1%
Between trials	2	5397·24	55·66	1%
Between orders	2	195·80	2·02	N.S.
Residual	42	96·97		
Total	53			

t, N_2O/oxy. = 6·805; 1%
t, N_2O/air = 3·842; 1%
t, oxy/air = 2·962; 1%

TABLE 22

Outline of analysis of variance of upper threshold scores

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between drugs	2	13466·80	38·61	1%
Between people	5	7678·46	22·02	1%
Between trials	2	7406·13	21·23	1%
Between orders	2	1234·02	3·53	5%
Residual	42	348·78		
Total	53			

t, N_2O/oxy = 8·470; 1%
t, N_2O/air = 6·265; 1%
t, oxy/air = 2·204; 5%

The results of the second part of the investigation were as follows: Air_1 75·50; Oxygen 73·83; N_2O 89·15; Air_2 79·52. Each value is expressed in msec and is based on 6 subjects for four, four eight and four trials.

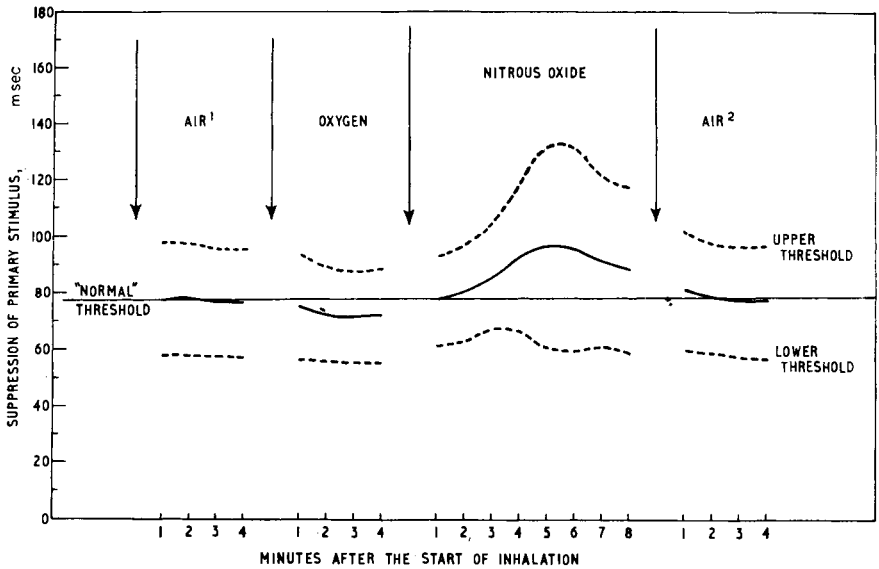


FIG. 10. The course of the four treatments on masking.

Fig. 10 shows graphically the course of the effect of the four treatments. The function presented is defined by the mean scores for the whole group. Differences between treatment means have been assessed by analysis of variance which is presented in Table 23.

TABLE 23

Outline of analysis of variance of mean threshold scores during the four treatments

Source	*D.F.*	*M.S.V.*	*F.*	*P.*
Between drugs	3	1560·57	17·28	1%
Between people	5	10997·36	121·74	1%
Residual	108	90·34		
Total	119			

t, air¹/oxy. = 1·338; 10%
t, air¹/N_2O = 4·905; 1%
t, air¹/air² = 0·736; N.S.
t, oxy/N_2O = 6·451; 1%
t, oxy:air² = 2·075; 5%
t, N_2O/air² = 4·013; 1%

DISCUSSION

The same pattern emerges from all the analyses, i.e. very significant F ratios are found for both individual differences, in *people* and for *treatments*. Two of the analyses have *order* effects at the 5% level which indicates that there is probably some carry over of effects from part to part of the test.

When the overall variance is broken down to give the individual comparisons, they show that the comparisons N_2O/air and N_2O/oxygen are always very significantly differentiated (i.e. 1%) but the comparison air/oxygen gives *t* values which lie at the 1% in two cases, the 5% in one case, and are not significant in one case.

The analysis of variance of the second part of the experiment shows a very significant overall variance despite the reduction in the concentration of N_2O. When the four treatments are broken down for the six possible comparisons the same pattern is seen, i.e. the comparison air (1 or 2)/N_2O is very significant as is the comparison oxygen/N_2O. The comparisons air/air and air (1 or 2)/oxygen are less significant although there is a tendency for oxygen to lead to a reduction in the interval of suppression. The composite curve for the group is presented in Fig 10 and indicates the slight effect of the oxygen treatment.

GENERAL SUMMARY OF EXPERIMENTS 5, 6 AND 7

The second part of this report is concerned with the description of three studies where pharmacological agents have been employed in order to vary the temporal aspects of masking in vision. Clearly, it is a shortcoming of the report that only one of the general aspects of the suppression of a primary stimulus has been investigated, but is is equally clear that the derivative drug postulate is not limited to temporal inhibition and its logic would apply equally to those aspects of masking denoted here as "spatial", "size", and "intensity".

The individual test hypotheses upon which experiments 5, 6 and 7 are based are, in turn, derived from the drug postulate (Eysenck, 1957). The drug postulate, itself derived from the Extraversion Theory, states that depressant drugs will increase inhibitory potentials and reduce excitation, whereas stimulant drugs will reduce inhibitory potentials and raise excitation. The chemical agents used: Sodium Amytal (a barbiturate and depressant), Meprobamate (an ataractic and mild depressant), Nitrous Oxide (a profound anaesthetic); and Dexedrine (an established stimulant), clearly fall into these categories. Placebo treatments have been used throughout as controls.

The observed results of the three investigations are largely in line with the stated prediction the exception being the failure to discriminate between Amytal and placebo in the first experiment, i.e. Experiment 5. Such "placebo effects", however, are to be excepted as "attitude" is said to influence masking, and they are, in this case, largely due to one subject (see Table 9, subject 2).

Experiment 6 produces the same lengthening of the masking interval due

to the meprobamate condition as that due to Amytal, except that in this case the meprobamate and placebo curves are clearly differentiated. In absolute values, the depression due to meprobamate appears smaller than that due to Amytal, but is comparable to it.

Divided into two parts, Experiment 7 is thought by the author to be of some interest. In the first part, what might be called the digital use of nitrous oxide, a very clear lengthening of the masking interval occurs with the N_2O treatment, and a smaller, but significant, shortening occurs with Oxygen. The method employed also indicates that the score designated "the interval of uncertainty" varies significantly with the treatment showing that the agent not only raises the threshold of masking, but also impairs discrimination. These aspects of the effects of N_2O are seen even more clearly in the second part of the gas experiments. (In particular, the reader is referred to Fig. 10.)

Fig. 10 and the data upon which it is based show clearly not only the changes in threshold but also the changes in discrimination as a function of time, outlining not only the slight improvement with pure Oxygen but also the gradual disintegration of discrimination which reaches its maximum after about $5^1/_2$ min from the onset of N_2O. There appears to be little, if any, effect during the first min to ninety sec. Another feature of interest in the time course experiment is the apparent adaptation to both Oxygen and N_2O, very slight in the former case and quite pronounced in the latter. Whether the inflexion point indicates some compensatory mechanism of the organism, which becomes operative as a counter to impairment (and the degree to which such compensation occurs), is speculative but possible and may justify further examination.

Any examination of the preceding experiments will indicate clearly that more than one explanation is applicable. The first four studies, concerned with the physical aspects of the masking phenomenon, do not further any resolution of the central-peripheral question, which is, of course, the cardinal point in the suggested explanatory hypothesis, despite the compelling parallel of masking and metacontrast. The dichoptic viewing studies of Kolers, as outlined above, suggest a non-peripheral explanation. It is unlikely, however, that any interocular effect could be considered conclusive to the problem of centrality (Day, 1958). On the other hand, the drug studies outlined in the later experiments (5, 6 and 7) in which the pharmacological agents employed have profoundly changed masking effects, strongly support a central explanation for two reasons: (a) the drugs used cannot be said to have an important direct effect upon the peripheral mechanisms; and (b) the drugs used were chosen as C.N.S. stimulants and depressants. The central action hypothesis is, of course, also supported by these later studies in that the specific test predictions are derived from the "drug postulate" which is itself a statement about central mediation. Finally, it must be considered that both types of experiment suggest the original hypothesis that, independent of locus, masking is due to inhibition.

The view taken by the author in this discussion is founded upon an analysis of the components of the visual stimulus in E and I type retinas advanced by Granit (1947). It may be remembered that Granit, on the basis of electro-

retinograms*, divided the total visual response into three separate components, the *a*, *b*, and *c* waves (page 50); each in turn roughly the recorded manifestation of processes defined as "*pre-excitatory inhibition*" (P 111), "*excitation*" (P 11)** and "*post-excitatory inhibition*" (P. 1), which fortunately describe as well as define the processes to which they are attached. In other words, the *a* wave (pre-excitatory inhibition) is a negative potential pulse which, at the optic nerve level, precedes the fundamental *b* wave, presumably reducing any existing "irrelevant" excitation in the same area. The *b* wave (excitation) is a positive pulse providing the neurological correlate of the stimulus, whereas the *c* wave (post-excitatory inhibition) is a wave which slowly swings to negative, probably as the result of preceding excitation.

From Granit's type of analysis, it can be reasonably argued that differences in the existing or generated levels of excitation inhibition in response to equivalent forms of visual stimulation should lead to differences in perception generally and, in this case, masking specifically. This is true particularly in the drug studies where there is a good theoretical basis predicting the increment or decrement of the excitation–inhibition balance which might be expected to subserve visual responses. It might be thought that this argument is highly speculative, linking together, as it does, a peripheral type inhibition only inferred from electroretinographic studies and the molar S–R inhibition which forms the growing point of Eysenck's theory of personality and the drug postulate. There are, however, several direct and indirect bases upon which such an inference might rest and from which the previously stated predictions might stem. These bases could be presented as follows:

(a) There is a direct source of evidence to be found in a study in which the extraversion (M.P.I.: E.) score of a group of 38 subjects was correlated with eight direct and derived scores of visual masking delineating the phenomenon from 5 to 70 msec. All eight coefficients are negative which is in the predicted direction, although none of them achieve significance. Clearly while it would be improper to infer too much from a series of insignificant relationships, the fact that each and every coefficient is as predicted by the original test hypothesis must lead to a probability which is, at least, suggestive.

(b) Indirectly, little doubt can be held that the effects of *pre-excitatory inhibition* transcend the retina and are influential in altering the optic nerve discharge, and, despite any modification which may occur due to the integrative action of this misnamed nerve, that the optic nerve discharge must have a cortical correlate. It is, therefore, a reasonable inference that *pre-* and *post-excitatory inhibition* is 'upstream' of the retina and has a cortical effect. If, as the derivative theory postu-

* Like Granit (1947) (p. 49) the author does not claim that the retinal components are the *causes* of excitation and inhibition, but they may well be measurable equivalents of these processes at higher levels of integration.

** "The size of the P 11 roughly reproduces something which one might, perhaps, call the 'intensity' or 'quantity' of the effect which is carried to the brain as a sensory message" (Granit, 1947, pp 82–83).

lates, there are differences in the central (cortical) processes which have been labelled "excitation" and "inhibition", then it seems a resonable assumption that not only should there be differences between people in the effects of the cortical correlates of processes initiated in the retina, but that these processes can be altered by centrally acting compounds which have been shown to change similar processes and functions in other modalities and in other tests.

(c) There is the known effect of centrally acting drugs upon sensory functions which may be determined by peripheral mechanisms, central mechanisms or, and this is probably nearer the truth, have components of both. The example which springs to mind immediately is critical flicker resolution, which may in some measure be determined by retinal processes of the type mentioned in this paper, but which is recommended as a method of standardizing the sedative effects of somnifacient drugs (i.e. central effects) (Landis, 1954). It is true that reactive inhibition theory has little to say about changes in c.f.f. thresholds, but, if the modification provided by satiation theory is accepted, certain predictions linking c.f.f. with central inhibition clearly follow.

(d) There is the semantic aspect of the many forms of inhibition, each predicating some decrement of performance contingent upon S–R connections, (i.e. work), or preceding excitation, (i.e. fatigue) (Duncan,1956). There seems every reason to believe that decrements in performance, though they differ in form, are examples of a basically similar process common to all neurologically mediated activities.

(e) Finally, there is the generally accepted scientific principle that similarities between phenomena are assumed until differences are demonstrated. For instance, most experimental hypotheses begin by asserting the null hypothesis, i.e. that two things are not different from each other, and demonstrating a refutation of the null hypothesis at some acceptable level of confidence. In the present case a direct comparison between "*central inhibition*", as defined by Eysenck, and "*pre- and post-excitatory inhibition*", as defined by Granit, cannot be made. It seems, however, legitimate to make the assumption that any neural correlates of the latter are influenced by drugs in the same way as are the former.

Even having accepted the assumption that there is sufficient similarity between I_R and *pre- and post-excitatory inhibition* to justify treating them as aspects of a basically similar process, it still remains to delineate precisely the relationship between the three recognisable components of the visual response and masking. The technique of visual masking is probably not appropriate to permit any definitive analysis, but the results do permit certain reasonable speculations. For instance, it is not unreasonable to assume that, for masking, the most important component, in Granit's terms, is *pre-excitatory* inhibition. This assumption would lead to conclusions which are not incompatible with the observed results, namely, that the straightforward inhibitory (pre-excitatory) pulse, which, like I_R differs from person to person, can like I_R, be increased by depressant drugs.

A second line of argument takes the view that masking is the result of the interaction of *post-excitatory inhibition* to the test stimulus and *pre-excitatory inhibition* to the masking stimulus, the two inhibitory components summating to reduce excitation from the test stimulus below some threshold of perception. Clearly, if the two forms of inhibition are of such a nature to permit addition, thc conclusions which must be drawn are again not inconsistent with the observed results. That is to say, there should be individual differences in the critical masking interval (as all the studies have shown) and that it could be expected that if depressant drugs not only increase the magnitude of I_R but also the *rate* at which it is generated then the degree of *post-excitatory inhibition* would be greater in the drug condition than otherwise. The obverse of this argument is, of course, true for the stimulant drug.

The third possibility would lie in some combination or interaction between the negative components and the "off" effect. Detailed consideration has not been given to the effect, although Granit suggests that the "off" response may augment the *a* wave. Apart from this, it is clear that both "on–off" and "off" effects must play an important role in vision by supplying a "changed state" signal against a "steady state" background and be important in the detection, if not the resolution, of contours by exaggerating the differences between patterns of excitation (Ratcliff *et al.*, 1958). Again, each of the explanations advanced permits the possibility that the components of the visual response are themselves composed of more than one source activity, which may be localised in either retinal or neural structures. On the available evidence, to site such loci in any precise structural position would be arbitrary.

SUMMARY

Some seven experiments have been conducted to determine a few of the parameters of the phenomenon of visual masking. The first four studies were concerned with the temporal, intensity, size and spatial aspects, whereas the second group of three experiments has been aimed at determining the effects of drugs which are mainly characterized by their action upon the central nervous system.

The investigation of the temporal aspects permits the generalization that between approximately 10 msec and the breakdown of masking, the critical masking interval, measured from the onset of the test stimulus to the onset of the masking stimulus, is virtually linear. No adequate explanation can be given for the failure of the masking function to remain linear below 10 msec. An earlier notion that this was due to a Bunsen–Roscoe effect is, however, questioned by investigations of intensity aspects.*

Reductions in the brightness of transmitted light by the use of filters lead to an inverse relationship with masking for the values tested. The explanation of this finding must, it is thought, lie in the reduced acuity

* The finding is, however, consistent with Granit's (1947) demonstration that the *a* (P 111) wave is reduced at low light intensities (pp. 52 and 113).

with reduced brightness or the reduced contrast between stimulus and background.

In an experiment designed to show the effects of masking when the two stimuli were spatially removed from each other, an approximately proportional relationship was established between the critical masking interval and the size of the separating gap.

There is a small but significant "size" effect which has emerged from two experiments. The annuli used cover a limited range of outer diameter sizes, but are sufficient to indicate that the larger they are the longer is the masking interval.

In the drug experiments the results have, on the whole, supported the derivative drug postulate and the inhibitory hypothesis by showing increases in the masking interval under depressant drug treatment and decreases under stimulant drugs. It is noteworthy also that discrimination, as characterized by the uncertainty interval, is also impaired under depressant drugs.

The explanation of the observed results is thought to lie in the similarities between the concept of "central inhibition" as described by Eysenck and the excitatory and inhibitory characteristics of the visual response, described by Granit and others.

Chapter 4

THE EFFECTS OF STIMULANT AND DEPRESSANT DRUGS ON AUDITORY CROSS MASKING

H. C. HOLLAND and JULIA BECERRA LURIA*

This investigation is concerned with an examination of the changes in auditory threshold in one ear when the opposite ear is being continuously stimulated by another sound. That such changes do occur has been demonstrated and the phenomenon has been called "cross masking". The phenomenon differs from straightforward auditory masking where the masking and test stimuli are administered to the same ear.

Although of interest to us because of the basic similarities the phenomenon of auditory cross masking has with other forms of masking in vision (see Chapter 3) and in kinaesthesis (Uttal 1961), the present study takes as its primary growing point the illuminating series of experiments conducted by Ingham (1957; 1959).

Earlier workers on auditory masking had asserted that there was no direct cross masking unless one referred to a very small effect which was thought to be due to "leakage" of sound from the stimulated to the non-stimulated ear. The results of Ingham's original experiments (Ingham, 1957) however, contradicted this view on two major points: (a) that (cross masking) occurs at intensities lower than those required for the *direct* stimulation of the opposite ear; and (b) cross masking varies with the frequency separation of the two tones. Ingham, *a propos* of this second point, however, draws attention to the fact, in his data, that whereas the masking effect falls off rapidly on either side of the masking frequency when this frequency is high, on the other hand when it is low, the generalization concerning spatial limitation does not seem to hold. For instance, he found (Ingham, 1959, Experiment II, Fig. 2) that with a masking tone of 200 cycles, the cross masking effect seemed to extend over the whole range of test frequencies, i.e. 170–3900 c/sec. This extended effect was not found when he used a masking tone of 1000 cycles, the effect being virtually limited to the two nearest test frequencies, i.e. 970 and 1030 c/sec.

In his discussion of his data, Ingham not only concludes that he has demonstrated that cross masking does exist, but advances four possible explanations for it at what amounts to a central, a peripheral and a conceptual level, calling these explanations the peripheral, the peripheral–central, the statistical and the central.

The first of these explanations, the peripheral, is mainly an analysis of the reasons why a purely peripheral explanation of the leakage of sound from one ear to the other must be excluded from consideration. Then follow three major explanations, the outline of each being briefly as follows:

* The writers are indebted to the Wallace Laboratories for support.

(a) *The peripheral–central explanation* is based on the protective reflex contractions of the tympanic muscles and the changing of the ears' "impedence". Ingham is, however, inclined to discount the reflex as the cause of the masking effect mainly on the evidence of previous work which has shown that the threshold for reflex contraction occurs at about 70–80 dB above the normal hearing threshold. (As the greatest pressure he used was 30 dB above threshold, he excluded this effect from masking). Ingham also considers the possibility of an inhibitory–facilitatory balance such as are found in visual structures and which have been advanced as an explanation of such phenomena as metacontrast and visual masking.

(b) The second explanation Ingham considers he calls the *statistical explanation*. The statistical explanation is couched in the language of communication theory and is based upon a modification of the noise-to-signal hypothesis. The explanation gets its name from the type of problem presented to the organism which compels it to decide whether an auditory event is a "signal" evoked by an external stimulus or part of the random variations composing the "noise". In Ingham's own words "..... do the two sets of activity rates come from the same population or not?"

(c) The final explanation, advanced by Ingham, he calls the *central explanation* and this is so called because it is based upon the possible "interaction occurring within the brain, a form of central inhibition". Although acknowledging the original postulate to Pavlov, Ingham then dissertates upon Broadbent's (1958) theory of stimulus competition with its concept of attention–inattention. He does not develop the explanation further, but in a later article (Ingham 1959) suggests that inhibition of neurones "..... need not be a direct effect between cells at the same level but that higher centres may inhibit the lower order neurones".

One aspect of auditory cross masking which Ingham touches on briefly, but which is the primary concern of this investigation, is that of individual differences. In his 1959 article (Experiment 1, Fig. 1) he outlines the masking effect for three groups, namely, two "neurotic" groups designated hysterics and disthymics and a normal group. Unfortunately the only statistic quoted for this study is that the F ratio for variations between frequencies for the neurotic group was significant at the 1% level.

The Present Investigation

The reader will be aware that we have presented Ingham's views at some length in the preceding paragraphs and it now behoves us to show the relevance of his work to the present study. Three reasons spring to mind immediately: (1) that the phenomenon described by Ingham is a form of masking and, as such, has special interest to the authors; (2), that Ingham has shown that individual differences in cross masking are demonstrable and may be relevant to personality classification (c/f Reed 1961); and

(3) the type of explanation advanced by him is similar to that within which other aspects of individual differences reported in this volume are evaluated. It is clear that the "central explanation" based upon a concept of central inhibition, despite the fact that it is illustrated by a rather specific example of a general inhibitory concept, is essentially the same as that used by Eysenck as the explanation of differences in the central mechanisms subserving the molar personality trait of extraversion–introversion (Eysenck 1953; 1957).

It is unnecessary to outline the detailed aspects of Eysenck's theory of personality either in form or derivation. As far as this investigation is concerned only one aspect need be stressed and that is the postulate which has been derived from the general theory and carries the assertion that stimulant drugs will increase excitation, decrease inhibition and have a characteristically introverting effect on the organism, whereas depressant drugs will decrease excitation, increase inhibition and have a characteristically extraverting effect. In a number of reported studies, the drug postulate of the extraversion theory has been used to predict the outcome of many psycho–pharmacological investigations employing conventional stimulant and depressant compounds as well as ataractics. In this study, the prediction is clear: *if cross masking is due to some aspect of central inhibition, and if central inhibition is increased by depressant drugs and reduced by stimulant drugs, then sodium amylobarbitone, being a depressant, should increase, and dexamphetamine sulphate, being a stimulant, should decrease the magnitude of cross masking.* This prediction was tested out by the following method.

Apparatus

Both masking and test tones were generated by an audiometer designed and constructed by Mr. J. Curtis at the Institute of Psychiatry. A fuller description is to appear in the future, but the following brief outline may be of assistance. (Reference may be made to block diagram – Fig. I).

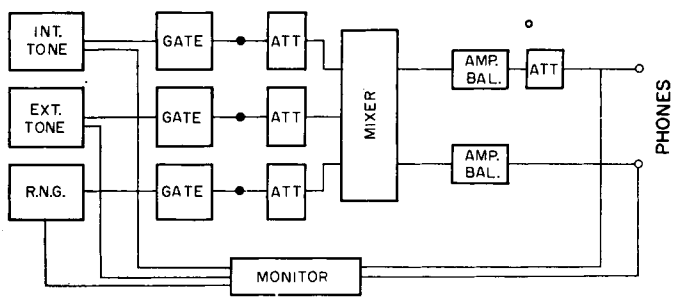


FIG. 1. Block diagram of the audiometer.

Both internal and external tones came from a Wein bridge oscillator generating pure sine waves continuously variable over the frequency range of 50c/sec–25Kc/sec. The instrument had facilities for random noise (Thyratron),

but this was not used in the present experiment. Tones were "gated" electronically, allowing rise and decay times of 0·1 and 0·07 sec respectively. Clickless switches prevented clicks at onset and offset of stimulation. Attenuators were fitted on all channels which permitted attenuation from −20 to+100 dB in 1 dB steps (±0·1 dB). Mixers allowed any combination of tones at any amplitude, over the range −20 – +100 dB, to be administered in any order or into either or both ears. Final amplification was by two stage gain, stabilized by Negative feedback. The output impedence was matched tothe 25 Ω earphones.

Subjects

A total of 12 subjects was used in the two experiments. They were of both sexes and their ages ranged from 19 to 38 years. Each subject was seen on 3 separate days under one of the three treatments; all were paid for their assistance.

Drugs

The drug treatments consisted of: (a) sodium amylobarbitone 290 mg. (b) dexamphetamine sulphate, 10 mg; and (c) a placebo (starch B.P.) The drugs were administered orally in identically appearing capsules, two to each day. One treatment was given each day, but not in all cases did the days run consecutively as in some cases the weekend intervened. An absorption period of 1 hour was allowed after taking each pair of capsules before any testing began. Drugs were administered by the double blind technique in accordance with a double 3×3 Latin square for each experiment.

Method

The testing procedure to be outlined took *in toto* about $1^1/_3$–$1^1/_2$ hours. This period being too long for a drugged subject to maintain concentration, it was consequently broken down into two test periods of 40–45 min with a 10–15 min rest interval between.

Two different masking tones were used, 400 c/sec and 1000 c/sec, one for each of two series. The test frequencies numbered ten, there being five to each series. In series I, (where the masking frequency was 400 c/sec), the five test frequencies were 170, 370, 600, 920 and 1250 c/sec. In series II, (masking frequency 1000 c/sec.) the test frequencies were 250, 450, 760, 1100 and 1400 c/sec. The order for the series and also the order in which the trials were given were randomized, the subject drawing a card on which one of the orders was printed. Having drawn this card on the first day the subject retained this order for subsequent sessions.

Sensitivity was assessed for each ear separately after a series of practice trials. The tone employed for assessing sensitivity was 1000 cycles. After sensitivity had been determined, the masking tone was presented to the more sensitive ear at 30 dB above the threshold of hearing. Test frequencies which were thus presented to the less sensitive ear, were assessed with the masking tone absent and present to the opposite ear. The difference between thresholds is the cross masking effect.

A method of limits was used to determine the threshold. Once the general sensitivity of the subject had been ascertained, the starting point for each threshold judgement was approximately 6 dB above or below the anticipated level. Changes in stimulus amplitude were in steps of 1 dB. There were four trials for each test frequency, two ascending and two descending, with a 15 sec rest between each trial. As far as is consistent with subjective judgement, the stimulus exposure was for periods of 5 sec.

The following outline is of the instructions given to the subject on the first day, and he was reminded of them on subsequent days. The room was sound proofed and the subject seated facing the wall with a small red light at about eye level. The headphones having been fitted, he was instructed as follows: (a) that the whole test period would be broken down into working periods and rest periods; and (b) that while the small red light in front of him (demonstration) was "on" he would be "working" and when off, "resting". Demonstration of the task took the following form: the specific test frequency was set to give an amplitude of some 50 dB. above threshold and the subject told that this was the tone he should "look" for through his left (or right) ear. The tone was administered several times in 5 sec "bleeps". It was then reduced to below threshold level and the subject then told that next time he heard it he was to tap on the arm of his chair with a pencil. When threshold was reached the amplitude was increased by about 6 dB and the subject again told to tap when he heard it — failure to tap was the signal in this case. This procedure was undergone again with the masking tone, the only extra instructions being, "I want you to disregard the tone in your other ear and tell me by tapping as soon as you hear the test tone."

Results

An outline of the results is presented first in tabular form in Table 1 which gives the means for the 12 subjects, for both series under the various treatment conditions. The values represented are also presented graphically in Fig. 2 and 3. In both cases the graphs indicate that maximum cross mask-

TABLE 1

Table of mean masking scores for the twelve subjects for the two series of test frequencies

	Sodium Amytal				
Series I	170	370	600	920	1250
400 c/sec.	0·94	5·42	1·69	2·82	1·56
Series II	250	430	760	1100	1400
1000 c/sec.	−0·90	1·75	1·07	2·56	1·79

	Dexedrine				
Series I	170	370	600	920	1250
400 c/sec.	0·77	5·94	0·50	2·81	0·75
Series II	250	430	760	1100	1400
1000 c/sec.	−0·58	−0·02	0·35	1·10	0·89
	Placebo				
Series I	170	370	600	920	1250
400 c/sec.	2·10	4·56	0·67	1·91	1·69
Series II	250	430	760	1100	1400
1000 c/sec.	0·11	1·27	0·48	2·60	1·24

ing occurs at those test frequencies nearest to the masking frequency.

Even a causal examination of Table 1 and Fig. 2 and 3 gives the impression that there are differences between drugs, although the differences between frequencies within drugs is also large and a large component due to individual differences should be considered — albeit not shown here. As a consequence of these several independent sources of variation, each set of scores for the two series were subjected to analysis of variance. The first analysis of variance is that for series I, where the masking tone was 400 cycles.

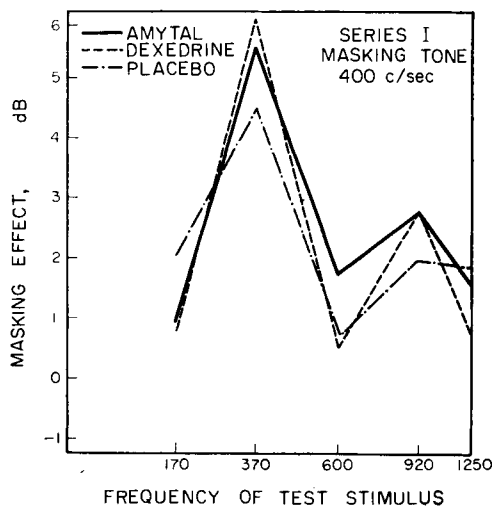


FIG. 2. Graph outlining the masking effect, for the various test frequencies and the three treatment conditions, in Series I.

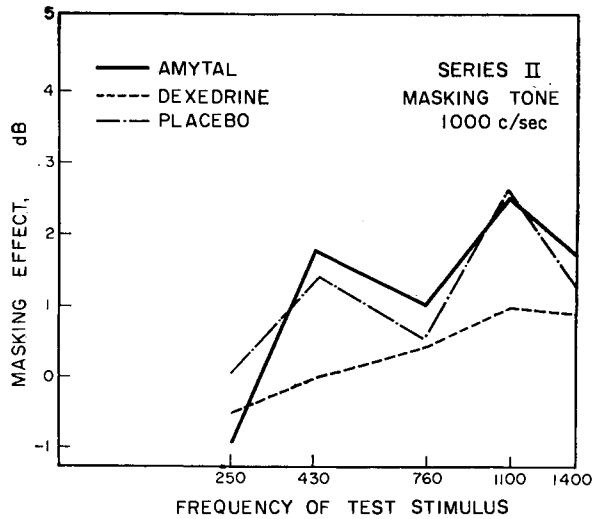


FIG. 3. Graph outlining the masking effect, for the various test frequencies and the three treatment conditions, in Series II.

TABLE 2

Series I. Masking tone = 400 c/sec

	Masking effect at frequencies				
	1 (170)	2 (370)	3 (600)	4 (920)	5 (1250)
Mean	1·27	5·31	0·95	2·51	1·33
	Masking effect due to drugs				
	(A) Amytal		(B) Dexedrine	(C) Placebo	
Mean	2·48		2·15	2·19	
	Outline of analysis of varience				
Source	D.F.	S.S.	M.S.V.	F.	P
Between Drugs	2	5·77	2·88	0·34	NS
Between Frequencies	4	463·91	115·98	13·84	1%
Between Subjects	11	247·73	22·52	2·69	1%
Residual	162	1357·16	8·38		
Total	179	2074·57			

T Tests between frequencies

$T_{12} = 6{\cdot}29$; 1% $T_{23} = 6{\cdot}39$; 1% $T_{34} = 2{\cdot}29$; 5% $T_{45} = 1{\cdot}73$; 10%

$T_{13} = 0{\cdot}46$; NS $T_{24} = 4{\cdot}11$; 1% $T_{35} = 0{\cdot}15$; NS

$T_{14} = 1{\cdot}81$; 10% $T_{25} = 5{\cdot}84$; 1%

$T_{15} = 0{\cdot}087$; NS

TABLE 3

Series II. Masking tone = 1000 c/sec

	Masking effect at frequencies				
	1 (250)	2 (430)	3 (760)	4 (1100)	5 (1400)
Mean	−0·46	1·00	0·63	2·09	1·31

	Masking effect due to drugs		
	(A) Amytal	(B) Dexedrine	(C) Placebo
Mean	1·25	0·34	1·14

	Outline of analysis of variance				
Source	D.F.	S.S.	M.S.V.	F.	P
Between drugs	2	29·34	14·67	1·98	NS
Between Frequencies	4	119·11	31·46	4·24	1%
Between Subjects	11	194·37	17·67	2·38	1%
Residual	162	1201·33	7·42		
Total	179	1550·87			

T Tests between frequencies

$T_{12} = 2{\cdot}27$; 5% $T_{23} = 0{\cdot}58$; NS $T_{34} = 2{\cdot}27$; 5% $T_{45} = 1{\cdot}21$; NS
$T_{13} = 1{\cdot}70$; 10% $T_{24} = 1{\cdot}70$; 10% $T_{35} = 1{\cdot}06$; NS
$T_{14} = 3{\cdot}97$; 1% $T_{25} = 0{\cdot}48$; NS
$T_{15} = 2{\cdot}76$; 1%

Examination of the masking means under the three treatments appears at first to be as would be predicted with the greatest masking under amytal, the least with dexedrine and with placebo lying midway between them. When analysed, however, by analysis of variance the observed differences do not differ at an acceptable level of confidence.

The variance between frequencies is highly significant as is the variance between subjects. A breakdown of this overall F ratio between frequencies, appended to the outline of the analysis of variance, is shown as several individual *t* tests, representing the separate comparisons between scores. On the whole, these *t* tests confirm earlier findings as shown by the cluster of significant values between frequency 2 (the nearest frequency to the masking frequency) and the others.

Inspection of the outline of series II where the masking tone was 1000 cycles again shows that the Amytal treatment session gives greatest masking,

with dexedrine least and placebo midway. When the overall components of variance are analysed for this series, the same pattern emerges as in the first but is less clear cut. This time the overall frequency variance gives only 2 out of 4 individual comparisons (between the closest frequency to the masking tone and the remaining test tones) as being clearly significant. The two comparisons between 4 and 2, 4 and 5, although in the right direction just fail at the acceptable level of 5%.

TABLE 4

Series I and II
Outline of analysis of variance

Source	*D.F.*	*S.S.*	*M.S.V.*	*F.*	*P*
Between Drugs	2	24·38	12·19	1·60	NS
Between Frequencies	9	756·21	84·02	11·00	1%
Between Subjects	11	274·51	24·96	3·27	1%
Between Series (masking tones)	1	166·48	166·48	21·79	1%
Residual	336	2570·35	7·64		
Total	359	3791·93			

Masking effect due to drugs (in dB)

(A)	(B)	(C)
Amytal	Dexedrine	Placebo
1·865	1·245	1·665

In the interest of completeness the two series have been combined into a single analysis which is outlined in Table 4. Again, however, the drug effect — the main experimental variable — is not significant. Of interest in this analysis is the source of variance between series which is very highly significant.

Interval of uncertainty scores

A number of studies conducted by one of us, in which drugs have been used have shown that depressant drugs tend to impair discrimination (Eysenck 1960). The impairment manifests itself as an increase in that interval between "certainty" and "uncertainty" which lies between an ascending and descending order of magnitude along psycho-physical continua. Elsewhere we have likened this to a hysteresis effect. It may well be due to some aspect of the "load" which the stimulus imposes on the resolving system and which is more influential when reducing magnitudes than when increasing them.

An alternative hypothesis is that effect of the drug is to increase the "noise" level of the system and that this increases the "noise-to-signal ratio" which is particularly relevant in the light of Ingham's "statistical explanation" of cross masking.

Unfortunately reference to Table 5 lends no support to this notion of impaired discrimination. The values presented indicate there is no difference between either the drug effect or the series effect.

TABLE 5

Table showing the interval of uncertainty scores under the three treatment conditions for both series*

	Series I	*Series II*	*Mean*
1. Amytal	1·75	1·79	1·769
2. Dexedrine	1.84	1.76	1·798
3. Placebo	2·02	1·77	1·895

Values represented are of the means of the separate frequency values

* Scores in dB.

Two supplementary examinations of the data were conducted. The first of these was to determine whether the masking effect increased during the testing session, due perhaps to some sort of fatigue effect. An "order" effect was consequently calculated but found to be not significant. The second examination was to ascertain, mainly for future reference rather than to draw any conclusions, whether there was any drug effect in masking at the frequency where maximum masking occurred. Two analyses of variance were conducted on the masking values observed at the 370 and 1100 cycle test frequencies. The result of these was to produce two non-significant F ratios, (370 cycles, $F = 0{\cdot}99$; 1100 cycles, $F = 1{\cdot}14$; with 2 and 22 degrees of freedom) for drug effects.

CONCLUSIONS

Like so many other investigations which use a hypothetico-deductive method, the observed results are at once both exciting and disappointing. On the positive side we see that testing has confirmed the earlier work of Ingham (if one accepts his exclusion of the head leakage hypothesis) that cross masking takes place. Figures 2 and 3 show clearly that the masking effect is limited to a narrow range close to the masking frequency; although the higher frequency (1000 c/sec) seems to be spread slightly. There is a tendency for masking values to be a little lower than those reported by Ingham.

Both the graphs, already referred to, are characterized by a second peak of masking value which very probably represents the effect of a second harmonic, in the case of the 400 cycle tone, and a sub-harmonic in the case of the 100 cycle tone.

One reason for the substantially smaller maximum masking with the 1000 cycle tone may be due, in part at least, to the further removal from it of the nearest test frequency, i.e. in the case of the 400 cycles, the maximum effect is noted only 30 cycles away at 370 c/sec, whereas the nearest frequency to the 1000 cycle tone is at 1100 c/sec, 100 cycles away. The apparently negative masking observed in several cases might represent a facilitatory effect.

On the debit side the main experimental hypothesis, that if the masking effect is due to central inhibition, it might be increased by depressant drugs, has not been confirmed at an acceptable level. The observed results, however, are such as to make it hard to deny that some tendency, as predicted, exists and might justify a re-examination.

Finally, an alternative appraisal of the results based upon a notion similar to Ingham's "statistical explanation" also failed to show the expected tendency.

Appendix, Part A

INTERRELATIONS BETWEEN TWO MEASURES OF PERCEPTUAL INHIBITION

H. C. HOLLAND and S. AIBA*

The attentive reader will have noticed that two reports in this volume are concerned with differing aspects of fundamentally the same problem, namely, suppression of the primary visual stimulus. The first of these is found in Chapter 2 , comprising an expansion and a modification of the phenomenon observed initially by Bidwell (1896) and is described as the simultaneous suppression of the primary of a pair of stimuli presented in immediate succession and the appearance of a hue complimentary to the original hue of the first stimulus. A lengthy description of the second phenomenon is found in Chapter 3 which is concerned with the original observation of Werner (1935) that the perception of a visually presented disk is suppressed if it is followed, within a critical interval, by an annulus whose inner diameter is the same size as the outer diameter of the disk and placed in the same phenomenal location.

Clearly the two phenomena in question have many differences and, although the explanatory concepts at which the authors arrive have certain elements in common, their detailed views diverge at several points. Nevertheless it has been felt that the similarities deserved closer investigation, and, as a first step it was decided to examine a group of 38 subjects, not originally investigated, as a joint venture but tested by both experimenters on the same day, for any evidence of intercorrelation between scores.

Bidwell's Phenomenon

As noted earlier, the phenomenon was first observed by Bidwell (1896, 1897) while investigating the problem of subjective colours. He found that when a disk such as that displayed in Fig. 1 Chapter 2 , was fully illuminated and rotated at a speed of approximately 5 rev/min a coloured stimulus seen through a cut-out sector of the disk was perceived as the complimentary colour rather than the original colour. This is a phenomenon which has caught the attention and interest of several authors and prompted them to study the effects of various drugs and various aspects of personality upon the effect (Lehman, 1950; Lehman and Csank, 1957; Levinson, 1952; Eysenck and Aiba, 1957; Kaplan, 1960a, 1960b). Definition of the phenomenon and the conditions under which it appears may be broadly stated as follows:

* The writers are indebted to the Wallace Laboratories for support of this investigation.

The stimulus condition may be defined as consisting of two separate flashes, the first one normally in colour and given for a relatively short period (5–50 msec) and the second being white and somewhat longer in duration and delivered immediately after the first.

The retinal area covered by the second flash is much larger than that of the original stimulus which is usually confined to the central fovea. Normally observation is made with the eye in various states of adaptation and with the luminance of both flashes at different photic levels. The suppression of the primary flash appears to be dependent upon the absolute intensity of the second stimulus compared to that of the first, assuming that the duration of both stimuli are constant. If, however, either intensity is reduced below a certain level, suppression does not take place and the phenomenon is not observed.

One of us (S.A.) recently made a detailed study of the Bidwell phenomenon and came to the conclusion that it is dependent upon the relative energies of the two flashes rather than their intensities if both stimuli do not exceed certain critical durations, as within these durations, a complete temporal summation of the energies of the light takes place in accordance with the Bunsen–Roscoe Law. If the durations are exceeded, the phenomenon becomes dependent upon the intensity of the second flash and not upon its duration. Another parameter was the state of light and dark adaptation of the eye, i.e. light adaptation increases the effective energy of the second flash, whereas dark adaptation reduces it. (See Chapter 2, for an elaboration of these findings.)

Werner's Phenomenon

The second phenomenon is one in which a disk (called the Primary Stimulus) is suppressed if it is followed within a critical time interval by an annulus (called the Secondary Stimulus), whose inner diameter is the same size as, and contiguous with, the disk, (Werner, 1940, 1945; Cheatham, 1952). In investigations conducted by one of us (H.C.H.) the period over which suppression is effective appears to be a function of, at least, the time between the onset of the first stimulus and the onset of the second. In a large group, (N = 83), the time interval was found to be 79·167 msec (S.D. 12·414), when the presentation times of both stimuli were 10 msec; and 17·397 msec (S.D. 10·881) when presentation times were increased to 70 msec. When the stimulus presentation times were changed at five levels from 10 to 70 msec, the interval over which suppression occurred was found to be virtually constant. In other investigations we have been able to demonstrate that drugs can significantly alter the interval over which the suppression effect takes place. (See Chapter 3 for an elaboration of these findings.)

*Apparatus (Bidwell)**

The apparatus consisted of two light sources, one providing illumination for the first (coloured) flash and the second providing the illumination

* Descriptions of this apparatus and that for producing the Werner phenomenon are presented in greater detail in Chapters 2 and 3, and abbreviated versions are given here for the sake of completeness.

for the second (white) flash. A rotating sectored disk controlled the duration of the flash. Light from a projector lamp was first diffused by a thin frosted glass and brought to a focus at the subject's eye by a lens after first passing through a red filter. The duration and frequency of the light were controlled by passing it through the sectored disk rotated at 15 rev/min. The front of the disk had a variable white sector which was illuminated by the second light source. A screen, which could be interposed between the disk and the subject'e eye had the dual purpose of occluding the light sources and acting as an adaptation field. The subject viewed the apparatus through a 3 mm artificial pupil. Illumination for the first source was varied by changes in the current supplied to the lamp (variac). Changes in the spectral quality of the light were minimal due to the use of the filter.

The method in the present study called for the following conditions:

(*a*) a red flash of 3·7 msec duration
(*b*) a white flash of either 63·1 msec.
(Exp. 1), or 126 msec (Exp. 2).

The luminance of the second flash was kept constant at 100 mL whereas the luminance of the first flash was the experimental variable.

Procedure

Following 10 min preliminary dark adaptation, the subject was light adapted to the adaptating field (4·85 mL) for a period of one minute through the artifical pupil. The adaptating field was then removed and the first stimulus sequence of 2·5 sec, dark–a red flash–a white flash, was delivered. At each repetition, the intensity of the red stimulus was increased in a step fashion. Between each sequence, there was an interval of 6 sec during which the adaptating field was viewed by the subject. The sequence continued until the threshold was reached, and this was defined as the point at which there were three consecutive reports of any red in the perception of the stimulus; (previously, of course, the subject had perceived the red stimulus as green). The descending threshold was the reverse, i.e. the sequences were repeated until the subject gave three consecutive reports of green whereas previously he had perceived stimuli which had contained decreasing proportions of red. The total procedure consisted of three ascending and three descending thresholds.

Apparatus (Werner)

The instrument used for this test was an electronic Dodge type tachistoscope with variable exposure and interval between exposures. The exposed fields were illuminated at 0·155 log. L by four 9 in. 6 W fluorescent tubes arranged around a rectangle and shaded to give a 7 in. square. The stimuli presented were a 17 mm black disk (vis. angle 0·913°) and a black annulus, with inner and outer diameters of 17 mm and 30 mm, (vis. angle 1·161°), both stimuli appearing on a white ground. The disk was viewed through, and the annulus reflected from, a half-silvered mirror placed at 45° to the line of regard.

Procedure

Testing was conducted in the dark. The subject was seated in front of the apparatus and fixated, binocularly, a red fixation point transmitted through a pin-hole in the centre of the annulus. He was then administered a sequence consisting of a disk (10 msec), an interval which was variable and an annulus (10 msec), and asked to report what he had seen. The interval was arranged so that he would see only an annulus. The sequence was administered again, but this time the interval was arranged so that a sequence of disk and annulus would be perceived. The subject was then asked after each presentation to report what had been seen and a series was then administered in which the interval between stimuli was varied in a 5 msec increasing order step fashion. When a report of the two stimuli was given on three consecutive occasions the lowest value of the three was defined as the threshold. The score was designated the ascending threshold. The descending threshold began with the clear perception of both stimuli, the interval being reduced until only the annulus was perceived.

Subjects

The subjects were 38 male apprentices whose ages ranged between 15 and 17 years. Their visual acuity and colour vision were within normal limits.

Scores

In all, 14 scores were derived from the joint investigation. Only 12 have, however, been used for correlational purposes; they were as follows:

(1) Bidwell Ascending threshold with second flash of 63·1 msec.
(2) ,, Descending threshold with second flash of 63·1 msec.
(3) ,, Threshold (ascending and descending) with second flash of 63·1 msec.
(4) ,, Ascending threshold with second flash of 126 msec.
(5) ,, Descending threshold with second flsah of 126 msec.
(6) ,, Threshold (ascending and descending) with second flash of 126 msec.
(7) Werner Interval between stimuli following exposure of both stimuli for 5 msec.
(8) ,, Interval between stimuli following exposure of both stimuli for 10 msec.
(9) ,, Interval between stimuli following exposure of both stimuli for 15 msec.
(10) ,, Interval between stimuli following exposure of both stimuli for 25 msec.
(11) ,, Interval between stimuli following exposure of both stimuli for 50 msec.
(12) Werner Interval between stimuli following exposure of both stimuli for 70 msec.

(13) „ "*Total*" is the sum of the intervals for trials 8–12.

(14) „ "*Slope*" is defined as trials $\frac{8+9}{2} - \frac{11+12}{2}$.

Prediction

The prediction for the experiment is fairly straightforward, namely:

(*a*) In the case of the Bidwell phenomenon, the inhibitory effect of the second (white) flash can be expressed as the degree to which the primary (red) flash must be increased in intensity to achieve primacy in perception.

(*b*) In the Werner effect both the primary (the disk) and the secondary (the annulus) stimulus are presented for the same duration at the same intensity. The score employed is the interval in msec over which suppression or inhibition of the first stimulus persists. If it is assumed that the length of the interval is some inhibitory function of the annulus, then the interval is proportional to inhibition.

Considered together, the joint predictions indicate that, despite the considerable differences which could be expected from these phenomena and the methods of their observation, the obtained scores would correlate.

Results

The means and standard deviations of all the scores are presented in Table 1.

TABLE 1

Table of means and standard deviations of measures

Investigation using a coloured primary stimulus			*Investigation using achromatic primary stimulus*		
Condition 1	mean	S.D.	Trials	mean	S.D.
Ascending threshold	79·55	8·62	5 msec	70·92	15·93
Descending threshold	75·03	7·69	10 msec	76·32	11·68
Threshold	77·21	7·48	15 msec	69·21	11·78
			25 msec	60·26	11·92
Condition 2			50 msec	33·71	10·56
Ascending threshold	93·73	9·01	70 msec	16·89	9·39
Descending threshold	90·26	7·54	"Total"	256·39	50·63
Threshold	91·60	8·54	"Slope"	47·72	7·87

For clarity three correlation matrices are presented in Tables 2,3 and 4. The first of these indicates the intercorrelation of scores for the Bidwell phenomenon, in which the primary stimulus is coloured. The second

TABLE 2

Table of intercorrelation of scores established using coloured primary stimulus

	1.	2.	3.	4.	5.	6.
1. Ascending threshold		0·855	0·964	0·844	0·562	0·768
2. Descending threshold			0·939	0·725	0·666	0·748
3. Threshold				0·813	0·611	0·774
4. Ascending threshold					0·788	0·921
5. Descending threshold						0·872
6. Threshold						

TABLE 3

Table of intercorrelation of scores established using achromatic primary stimulus

	1.	2.	3.	4.	5.	6.	7.	8.
1. 5 msec		0·786	0·740	0·716	0·603	0·501	0·741	0·442
2. 10 msec			0·954	0·943	0·694	0·682	0·946	0·609
3. 15 msec				0·948	0·712	0·697	0·954	0·579
4. 25 msec					0·756	0·725	0·966	0·497
5. 50 msec						0·757	0·853	−0·039
6. 70 msec							0·834	−0·072
7. "Total"								0·371
8. "Slope"								–

N = 38
r 0·325 = 5%
0·418 = 1%

matrix displays the correlation coefficients of the Werner phenomenon including the individual trial scores, plus the two scores "total" and "slope". In this case the stimuli were achromatic. The third matrix is of the combined Bidwell and Werner scores with the exception of the two derived scores to which reference has already been made. The factorial composition of the test was assessed by a Thurstone Complete Centroid analysis. The matrix of twelve variables shown in Table 4 was factor analysed and four factors extracted, accounting for 45, 30, 5 and 3 per cent of the total variance. Because of the distribution of test variables, following a single rotation (see Fig. 1 and Table 5), factor 1 was indentified as a Bidwell test factor and factor 2 as a Werner test factor. The remaining factors were considered as residuals.

TABLE 4

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Ascending threshold (Bidwell)		0·955	0·964	0·844	0·562	0·768	0·052	0·165	0·161	0·220	0·127	0·346
2. Descending threshold (Bidwell)			0·939	0·725	0·666	0·748	0·124	0·186	0·186	0·253	0·205	0·345
3. Threshold (Bidwell)				0·813	0·611	0·774	0·083	0·205	0·200	0·259	0·158	0·347
4. Ascending threshold (Bidwell)					0·788	0·921	0·060	0·111	0·090	0·149	0·225	0·407
5. Descending threshold (Bidwell)						0·872	0·088	0·086	0·082	0·158	0·246	0·428
6. Threshold (Bidwell)							0·090	0·121	0·142	0·190	0·200	0·410
7. 5 msec (Werner)								0·786	0·740	0·716	0·603	0·501
8. 10 msec (Werner)									0·954	0·943	0·694	0·682
9. 15 msec (Werner)										0·948	0·712	0·697
10. 25 msec (Werner)											0·756	0·725
11. 50 msec (Wenrer)												0·757
12. 70 msec (Werner)												–

N = 38

r = 0,325 = 5%
r = 0·418 = 1%

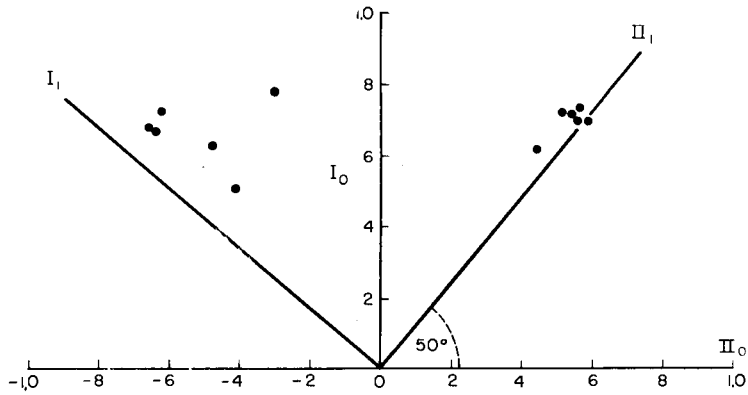


FIG. 1. Plot of original and rotated factor loadings.

TABLE 5

Original and rotated factor loadings of Bidwell and Werner test variables

	I_0	II_0	I_1	II_1	h^2
1.	0·697	0·572	0·010	0·902	0·813
2.	0·723	0·524	0·064	0·891	0·797
3.	0·726	0·546	0·049	0·907	0·825
4.	0·693	0·576	0·005	0·901	0·812
5.	0·613	0·442	0·055	0·754	0·571
6.	0·708	0·556	0·029	0·900	0·810
7.	0·506	−0·545	0·742	0·038	0·553
8.	0·663	−0·649	0·923	0·091	0·861
9.	0·660	−0·650	0·922	0·088	0·858
10.	0·720	−0·605	0·926	0·163	0·884
11.	0·626	−0·484	0·774	0·169	0·626
12.	0·770	−0·299	0·724	0·398	0·682

DISCUSSION OF RESULTS

The main points of interest in the present investigation are outlined in the third matrix (Table 4). The first examination of this matrix indicates that the different threshold and trial scores are strongly related to each other, whereas, with one exception, there are no significant correlations between the tests. The exception is the series of significant correlations between the 70 msec trial on the Werner phenomenon and the Bidwell trials; all of them lying between the 5% and 1% levels. It is also of interest to note that the remaining correlations of the Bidwell–Werner trials are all positive, and there is a tendency for them to increase in value as the Werner trials increase in duration of the stimuli.

Why the correlation between scores should increase with the increasing durations of the Werner effect, we cannot explain. However, the explanation may lie in the intervals between stimuli. It may be remembered that in the Bidwell phenomenon, the second flash follows immediately after the first and that in the Werner effect, the interval becomes progressively shorter, as the duration of the stimuli is increased. An example is seen in the longest duration of the Werner effect trials, where the interval between stimuli is only 16·89 msec.

CONCLUSIONS

Two phenomena, the Bidwell and Werner effects, were given to the same group of 38 subjects. Both phenomena involve the suppression of the primary of a sequence of two stimuli, the Bidwell suppressing a chromatic stimulus and inducing the complimentary hue and the Werner suppressing a disk which is followed by an annulus. The scores obtained are thought to represent the inhibitory function of the second stimulus in each case. Intercorrelation of scores on the several measures indicate slight relationship until the interval between stimuli on the Werner effect approaches the conditions of the Bidwell phenomenon. Within each test, the trials and conditions are highly intercorrelated. The factor analysis has simply emphasized the earlier visual examination of the matrix showing two clear test factors which are almost completely orthogonal to each other altough there is again the tendency for the correlation between the factors to increase as the interval between stimuli on the Werner test approaches the immediate succession of the Bidwell conditions.

Chapter 5

EXCITATION–INHIBITION AND THE THEORY OF NEUROSIS: A STUDY OF THE SEDATION THRESHOLD

G. S. CLARIDGE and R. N. HERRINGTON*

Undoubtedly one of the most important contributions psycho-pharmacology can make to general psychology is the investigation of individual variations in the response to drugs. Commonly observed in everyday life, in experimental situations these individual differences have typically appeared as variations in the effect of a standard dose of a particular drug on the efficiency of some kind of performance. A recent example of this sort of finding has appeared in an investigation reported by Drew *et al.* (1959), who noted individual differences in the degree to which alcohol impaired performance on a simulated driving task. It was concluded that these variations in response to the drug were related mainly to personality factors, particularly with regard to extraversion.

Exactly 30 years before the publication of that report, McDougall (1929) proposed a chemical theory of introversion–extraversion which took account of the greater susceptibility of the extravert to the effects of alcohol and similar substances. Later (1942), Sheldon and Stevens emphasized the resistance to alcohol of "cerebrotonic" individuals, who, they proposed, were characterized behaviourally by emotional tension and physiologically by strong control of cortical over visceral and somatic functions. Meanwhile, Pavlov (1934) had noted that dogs of various temperamental types differed in their drug sensitivity and towards the end of World War II the implications of Pavlov's findings for the treatment of battle neuroses were recognized by Sargant and Shorvon (1945). However, in the subsequent 10 years or so their application in clinical practice was based largely on unsystematic observation. Even as recently as 1956, Sargant and Slater, in discussing chemical sedation in psychiatry, noted:

* The authors wish to express their thanks to Brigadier J. McGhie, formerly Commanding Officer at the Royal Victoria Hospital, Netley, for his interest and co-operation at all stages of the work reported in this paper. To other colleagues, past and present, at the hospital goes our gratitude for making their patients available for testing. We also thank Professor Eysenck for his encouragement of the line of research developed and for his critical reading of the manuscript. The work of one of us (G.S. Claridge) was made possible by a grant from the US Army (Grant No: Da-91-591-EUC-1458), to whom acknowledgment is made. Dr. Claridge is now Director of the Department of Experimental and Clinical Psychology at Barrow Hospital, and Dr. Herrington is M.R.C. Research Fellow, Physiology Dept., Aberdeen University.

"Another principle which has in practice received but scant consideration is that of individual variability. Probably many drugs would be more effectively used in treatment if more attention were paid to individual susceptibility.... Human (beings like Pavlov's animals) show just as much variation from one to another, though as yet we know too little to guide us surely in varying the doses of drugs to suit each case."

Thus, even though it has long been suspected that there is a link between personality and drug tolerance, it is, in fact, only very recently that research workers in human psychology have been attracted to the problem sufficiently for them to consider investigating it experimentally.

A major contribution to this field, both conceptually and experimentally, has been made by Eysenck, who has based his theorizing partly on Pavlov, while at the same time attempting to bring McDougall's theory more in line with modern psychological thinking. Eysenck has suggested (1955) that there is a natural variation among individuals in the state of balance between central excitatory and inhibitory processes and that this is reflected behaviourally in varying degrees of extraversion. Thus, the introvert is said to be characterized by a predominance of excitatory potential, developing inhibition only weakly, while the extravert builds up inhibition rapidly, showing low levels of excitation. A second postulate (1957a) links this theory of personality with the theory of drug action, since it is proposed that control of the excitation–inhibition balance is possible by means of depressant and stimulant drugs. The suggestion is that depressant drugs decrease excitation and increase inhibition, while the opposite effect is produced by stimulant drugs.

General confirmation of this theory is found in the tendency for the changes in performance induced by stimulant and depressant drugs to be in the direction of the kind of performance found, respectively, in introverts and extraverts. Thus, the demonstration that introverts condition better than extraverts (Franks 1957) is paralleled by the finding that Dexedrine facilitates and sodium amytal retards conditioning (Franks and Laverty, 1955; Franks and Trouton, 1958). It is with investigations of the kind reported by Franks that most of those working within Eysenck's theory have been concerned (e.g. Eysenck and Easterbrook, 1960). The most obvious way of testing the theory is, of course, to compare the effects of stimulant and depressant drugs on measures which are known to discriminate introverts and extraverts.

Another approach to the problem, however, was pointed out by Eysenck himself in his original statement of the drug postulate (1957). If it is true that individuals vary in their position on the continuum of excitation–inhibition, then it should be possible to measure the amount of a depressant or stimulant drug required by various individuals to reach a criterion of inhibition or excitation. Thus, the extravert, being already basically high in inhibition, should, compared with the introvert, require a smaller dose of, say, alcohol to reach a given level of intoxication.

This fact, already well-known to most drinking laymen, had not been investigated experimentally until, contemporaneously with Eysenck's prediction, a paper was published by Shagass (1954), who, working independently, had discovered a method of measuring individual differences in what he called the "sedation threshold". He defined this in terms of the amount of sodium amytal required to bring about certain behavioural and other changes in the individual, the threshold itself lying somewhere between the state of complete wakefulness and that of complete sedation. Two methods of determining the threshold were employed by Shagass. First, the point of onset of slurred speech gave an approximate estimate of its position. Secondly, a more accurate determination was made by means of EEG changes, specifically the point at which inflexion occurs in the amplitude curve of induced fast frontal activity.

Shagass, at that stage concerned mainly with the differentiation of neurosis and pseudoneurotic schizophrenia, considered that individual differences in the tolerance of sodium amytal were related to variations in the degree of tension found in psychiatric patients, a correlation being reported between a rating of this symptom and sedation threshold. This interpretation was later revised when Shagass and Naiman reported that sedation threshold was correlated with the degree of manifest anxiety, rather than tension, a relationship shown by these authors to obtain in both normals (1955) and neurotics (1956). In the latter study, Shagass and Naiman also demonstrated that various types of neurotic could be discriminated by means of the sedation threshold, anxiety states having high and hysterics low thresholds. This finding was later confirmed on a much larger group by Shagass and Jones (1958), who again embraced the hypothesis that the sedation threshold is related to manifest anxiety, the assumption being that the hysteric displays less manifest anxiety than the dysthymic. In the same paper, however, Shagass and Jones also consider another possible interpretation of the results, viz. that sedation threshold is positively related to the degree of obsessionality. The authors assume a continuum of neurotic personality ranging from obsessionality to hysteria, relating this to the introversion–extraversion dimension of Eysenck. They explain this dual interpretation of their results by suggesting that there are two mechanisms mediating anxiety, the sedation threshold reflecting the "degree of activity of that neurophysiological mechanism of anxiety which predominates in obsessional personalities".

The relevance of introversion–extraversion was later made more explicit by Shagass and Kerenyi (1958a) who report a positive correlation between sedation threshold and a measure of introversion. At the same time, they retain the interpretation that manifest anxiety is also an important determinant of tolerance of depressant drugs and restate their dual mechanism theory of anxiety. This insistence by Shagass and his colleagues on the role played by anxiety in neurosis is of particular interest here because of its relevance to the theme to be developed in this paper. In order to see how the present authors' approach has evolved as a result of experimental investigation in this field, it is necessary first to return to a reconsideration of Eysenck's theory.

It is clear that the findings of Shagass and his colleagues, on the whole, point to the essential correctness of Eysenck's theoretical position. This is true in two respects. First, the correlation, reported in the paper with Naiman, between sedation threshold and introversion directly corroborates the prediction made by Eysenck from his twin postulates about personality and drug action. Secondly, the differences found between neurotic groups on sedation threshold are in keeping with Eysenck's analysis of neurosis (1947), since in his work he has followed the Jungian view that the hysteric is characteristically extraverted and the dysthymic characteristically introverted. In terms of Eysenck's later theorizing about personality (1957b) this means that hysterics and dysthymics should fall at opposite ends of the excitation–inhibition continuum, with normals occupying an intermediate position. Shown by Franks to be true for conditioning (1956), this hypothesis has been confirmed more recently by Claridge (1960). Here a variety of tests was investigated, including measures of spiral aftereffect, pursuit rotor performance, time error, and time judgment. Hysterics showed consistent evidence of greater susceptibility to inhibition than dysthymics, while in most cases, normals fell between the two neurotic groups. These findings later received further support from the results of a canonical variate analysis carried out by S.B.G. Eysenck *et al.* (1960) on 10 selected measures from the investigation by Claridge.

Despite these results, however, there was evidence from the original data of the study that the relationship between introversion–extraversion and dysthymia–hysteria was more complex than Eysenck's dimensional analysis had suggested. For example, in terms of the extraversion scale of the Maudsley Personality Inventory, hysterics, although more extraverted than dysthymics, were somewhat *less* extraverted than normals. This tendency, which had already been reported by Sigal *et al.* (1958), has consistently appeared since then in the writers' work. The position would seem to be then, that, while the dimension of dysthymia–hysteria has perfect or near-perfect identity with the underlying continuum of excitation–inhibition, neither of these has perfect identity with the introversion–extraversion dimension*. For this reason, the hysteric tends to show greater inhibition than normals of equivalent extraversion and the dysthymic greater excitation than equally introverted normals.

In order to see what variables additional to extraversion had influenced the performance of dysthymics and hysterics, a factor analysis was carried out by Claridge on 30 measures from his original study cited above. In addition to extraversion, a factor was extracted which was identifiable as

* Part of this fact has already been recognised in a recent publication by Eysenck (1960b) who says, "In spite of this observed relationship between extraversion–introversion, on the one hand, and dysthymia–hysteria, on the other, it should not be assumed that any perfect identity is postulated between the two dimensions. The events which are responsible for the neurotic breakdown... must in part determine the form which the symptom shall take". We would perhaps go further than this in suggesting that the form the symptom takes may itself influence the relationship between the two behavioural dimensions and the common underlying process of excitation–inhibition. This is, however, anticipating a point which is developed in the main body of this paper.

one of drive. High loadings on this factor of measures, such as pursuit rotor performance level, lent some support to this interpretation, since learning theorists such as Hull (1943) have traditionally regarded speed or level of performance as partly a function of positive drive level. A later test of this was made by Claridge (1961) using a serial reaction time task, where the effects on the measures taken of such variables as habit and inhibition could be eliminated. Supporting the hypothesis that there were group differences in drive, it was found that dysthymics performed at a faster rate than normals, who were in turn faster than hysterics.

In what sense the factor derived from the study of dysthymia–hysteria could be regarded as one of drive was indicated by a positive loading on the factor of the neuroticism scale of the Maudsley Personality Inventory. This is significant in two respects. First, it clearly reflects the tendency, found both in the study itself and by Sigal *et al. (op. cit.)* for hysterics to have lower scores on this scale than dysthymics. Since neuroticism has been regarded (Eysenck, 1955) as a form of drive related to autonomic excitability, the finding suggests a difference in this respect between the two types of neurotic. Secondly, supporting this interpretation are the respective clinical descriptions of hysterics and dysthymics. While the dysthymic is characterized by a heightened level of manifest anxiety, the hysteric is said to "convert" his anxiety into bodily symptoms, thus reducing anxiety even, in some cases, to the abnormally low level reflected clinically in the so-called "belle indifference".

With these facts in mind, an hypothesis was formulated to account for the disproportionate excitatory and inhibitory effects seen in dysthymics and hysterics. It was suggested that changes in autonomic drive at the onset of neurotic breakdown resulted in a shift in the excitation–inhibition balance assumed to underly introversion–extraversion. The heightened anxiety found in dysthymics was said to result, in these patients, in an increase in excitation and a decrease in the proneness to inhibition. A corresponding increase in inhibition and a lowering of excitation level was assumed to occur in hysterics.

Although only informally recognized at that stage, it was clear even then that current arousal theories of behaviour could probably contribute greatly to an understanding of the mechanisms whereby drive could influence the excitation–inhibition balance in the manner suggested. Particularly revelant was Hebb's (1955) formulation of drive as an arousal process arising from the activity of the ascending brain stem reticular formation. This conception of drive as a central process having activating excitatory properties suggested that the level of arousal may be one, at least, of the influences maintaining the cortical excitation–inhibition balance visualized by Eysenck. Changes, in neurotics, in the level of arousal or drive would then be an important factor affecting their respective performances on objective tests of the kind used to study the inhibition theory of introversion–extraversion.

As will become clear later, this theory was eventually elaborated and its implications considered in more detail. This occurred as the result of two further investigations of dysthymia–hysteria. The first of these was carried

out because the need was felt for an independent measure of the state of arousal and/or excitation–inhibition in neurotics and for further information about the relationship of this measure to other kinds of performance. Consideration of the literature indicated that the sedation threshold technique was ideally suited to this purpose, for two reasons. First, as we have seen, Eysenck himself has considered the sedation threshold a direct index of the excitation–inhibition balance, principally because of its relationship with extraversion. Secondly, Shagass, while confirming this, has at the same time felt the need to reconcile the finding with that of a simultaneous relationship with anxiety. This emphasis by Shagass on both introversion and anxiety thus coincided closely with our own hypothesis which, it is interesting to note, had been developed as the result of an entirely different approach to the problems of neurosis.

The first study to be described has already been reported in more detail by Claridge and Herrington (1960), who experimented with a new technique for measuring the sedation threshold. This technique and its application to the problems under discussion will be briefly outlined in the next section. Following an appraisal of these results a further investigation of the sedation threshold, not previously reported, will be described and an interpretation of all the results presented.

SEDATION THRESHOLD, PERSONALITY, AND PERFORMANCE

Despite the success reported by Shagass and his colleagues, attempts by other workers to duplicate his methods have given rise to conflicting opinions about the relative merits of slurred speech and EEG changes as indices of the sedation threshold. Of those using an EEG index, a Danish worker, Nymgaard (1959), has reported positive findings, while Seager (1959, 1960), and Bradley and Jeavons (1957), with a modification of Shagass' original technique, were also able to make satisfactory and reproducible measurements of sedation threshold. However, two other workers, Roberts (1959), and Kawi (1958), rejected this method in favour of slurred speech. Also using slurred speech, Laverty (1958) was able to differentiate extraverted and introverted neurotics and normals and to demonstrate a slight increase in extraversion under sodium amytal. At the same time, he does report, in common with other workers using this method, that it was difficult in many cases to assess the sedation threshold accurately. Much more critical are Thorpe and Barker (1957), who, following an attempt to assess the objectivity of Shagass' methods, conclude that the onset of slurred speech is quite unsatisfactory as an indicator of the sedation threshold. Agreeing with these authors, Boudreau (1958) elected to use the EEG method, but found it possible to estimate a threshold in only about half his cases. An even higher rejection rate is reported by Ackner and Pampiglione (1958), who used both slurred speech and EEG changes. They found that it was difficult, or even impossible, to determine a threshold in at least two-thirds of their patients.

Attempts to assess sedation threshold by some other method have been few.* Fink (1958) reports some success using as an index the presence of lateral gaze nystagmus, while Shagass and Kerenyi (1958b) have measured the sleep threshold in terms of unresponsiveness to verbal stimuli. Both of these are said to give results comparable with those obtained by the original methods.

Equally conflicting opinions are found in the papers quoted about the relationship between sedation threshold (where it could be assessed) and personality. In the study by Ackner and Pampiglione *(op. cit.)*, there appeared to be no relationship between sedation threshold and rated anxiety, nor did hysterics and dysthymics differ on the measure. On the other hand, Boudreau *(op. cit.)* using small numbers, found group differences in sedation threshold comparable with those of Shagass, although he agrees with Seager *(op. cit.)* in finding no correlation with clinically rated anxiety. Kawi *(op. cit.)*, however, reports that clinically judged anxiety *does* correlate significantly with sedation threshold for both sodium amytal and ethyl alcohol. A questionnaire measure of anxiety — the MAS — did not correlate with the threshold for amytal, but did where alcohol was used.

It seems very likely that the discrepancies in the findings of these various authors are due mainly to the unreliability of the sedation threshold techniques used. With this in mind, the authors set out to devise a new method of measuring the sedation threshold.** It was clear that the technique would need to be more reliable and more objective than slurred speech, which appears too inconsistently to be regarded as a useful index. On the other hand, it was desirable that it should be easier to administer than is the case with an EEG method, which, while more satisfactory than slurred speech, requires more complex apparatus than its reliability would seem to warrant.

The procedure followed was to assess sedation threshold in terms of the effect of sodium amytal on a simple task of attention. The stimulus material consisted of a tape-recording of random digits. While receiving a continuous intravenous infusion of sodium amytal at the rate of 0·1 g/min, the subject

* Since writing this paper the authors' attention has been drawn to some recent work at the University of North Carolina (Perez-Reyes, personal communication), where the sedation threshold has apparently been successfully determined in terms of the inhibition of the GSR by pentothal anaesthesia. It is further interesting to note that the theoretical interpretation given by Perez-Reyes (1960) coincides very closely with that developed here. He has, for example, suggested that the effect of barbiturates is to disturb the balance between excitatory and inhibitory processes and has discussed some of the possible neurophysiological mechanisms underlying excitation–inhibition.

** It may be objected that the method to be outlined measures not the sedation threshold, but the sleep threshold described by Shagass and Kerenyi (1958b). While it is true that, in all cases, a greater absolute amount of drug is required to reach the criterion used for a threshold, the authors prefer to retain the term originally introduced by Shagass. This is partly because the term "sedation threshold" is probably more familiar to the reader, but also partly because the technique to be described does not necessarily induce a depth of sedation leading to sleep. Of course, we must not overlook the fact that the difficulties of defining early but consistent effects of the drug may *ipso facto* account for the unreliability of previous attempts to find a threshold of "sedation". While these difficulties may eventually be overcome by more refined techniques, for the present, it seems desirable, in the interests of reliability, to err on the side of "over-dosage".

was required to respond by doubling the digits, which occurred regularly at intervals of 2 sec.

The digits were grouped on a score sheet into blocks of five, a record being made of the number of errors made in each block. The threshold was taken as the point midway between the last two blocks with less than 50 per cent error and the first two blocks in which errors exceeded 50 per cent. In the majority of cases these blocks were consecutive. The amount of drug administered at this point was determined from a chart relating blocks to drug received and this dosage corrected for the weight of the patient, giving the threshold in terms of mg/kg.

During the early stages of using this technique each subject's performance was individually plotted in order to facilitate the estimation of his threshold. This gave graphs typically of the form shown in Fig. 1. Later, however, as the reliability of the method proved itself, it became clear that the thresholds could be easily read from the original score sheets.

Continuous experience with this technique over, at the time of writing, a period of more than a year has shown it to be even more satisfactory than was hoped. Requiring little apparatus, the method may be easily duplicated by other workers and, in addition to its use as a research tool, for which purpose it was originally designed, it has proved helpful to the clinician as an aid to diagnosis.

TABLE 1

Mean sedation threshold (in mg/kg) for each group

	Dysthymics	*Normals*	*Hysterics*
Mean	10·18	7·86	6·43
SD	1·608	1·313	1·774

F-ratio: 21·837, $p < 0{\cdot}001$

t-tests: Dysthymics v. Hysterics, 6·544, $p < 0{\cdot}001$; Dysthymics v. Normals, 4·049, $p < 0{\cdot}001$; Hysterics v. Normals, 2·496, $p < 0{\cdot}02$.

The technique was first used with three groups of subjects, viz. dysthymics, hysterics, and normals, there being 16 subjects in each group. As expected from the work reviewed in the previous section and as can be seen from Table I and Fig. 1 dysthymics had higher thresholds than normals, who in turn had higher thresholds than hysterics. All group comparisons were significant.

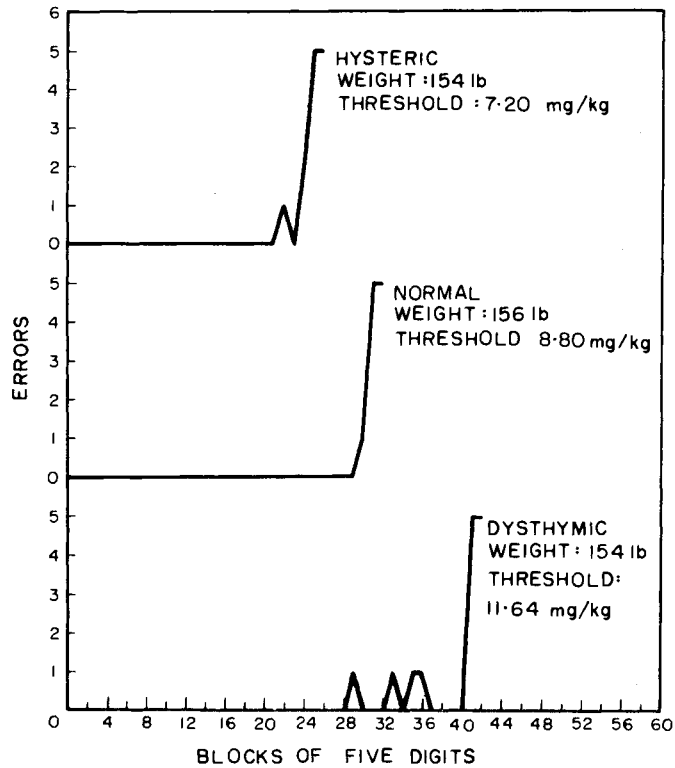


FIG. 1. Typical sedation threshold curves: one subject from each group.

Our main interest, however, lay not so much in the group differences as in the relationships within the total sample between sedation threshold and measures of (a) performance and (b) personality. The first of these was examined by giving each subject two objective performance tests. One of these was the serial reaction time task previously used with psychotics by Venables (1959), and later with neurotics by Claridge (1961). Briefly the test consists of a display panel of 5 lights and a set of 5 keys. In operation, the subject presses the key appropriate to the light which is on at any one time. This response is instrumental in illuminating another light to which a further response is made, and so on.

Several measures from this test were correlated with sedation threshold. It was found that the index of speed, used in the study described earlier to differentiate hysterics and dysthymics, correlated 0·366 ($p < 0·05$) with sedation threshold. On the other hand, the conventional measures of inhibition in terms of rate of decline in speed and reminiscence failed to correlate with sedation threshold, although the latter did correlate significantly with errors on the test to the extent of —0·395 ($p < 0·01$). Interpre-

tation of these correlations was more complex than had been anticipated and for a detailed account of the argument relating to them, the reader is referred to the original publication. Briefly, it was suggested that performance on the serial reaction time test was determined by the tendency on the part of all subjects to maintain an equilibrium between excitatory and inhibitory processes. If it is assumed, as we have here, that changes in arousal or drive influence the subject's proneness to inhibition, then the excitatory (arousal) factor, which both speed and sedation threshold have in common, must have allowed the subject with high drive (the dysthymic) to make a greater number of responses on the task without accumulating any more inhibition than the subject with low drive (the hysteric). Hence the zero correlation between threshold and both rate of decline and reminiscence. The significant correlation with errors is explicable if one assumes that the committing of errors is one way of partly dissipating inhibition as the task proceeds and helping to maintain the excitation–inhibition balance. One would then expect that the subject with low drive, and therefore greater proneness to inhibition, would need to commit more errors than the highly driven individual with weak inhibitory tendencies.

There was certainly some evidence, therefore, that the sedation threshold was some kind of measure of the balance between excitatory and inhibitory processes and that our introduction of arousal or drive as a factor influencing this had not been entirely incorrect. This point will emerge even more clearly when the relationships with personality are considered.

In the meantime, however, mention should be made of the relationship between sedation threshold and performance on another objective test, viz. the Archimedes spiral. This finding is of interest, not because of its direct bearing on the argument being developed here, but because of the close relationship found between the two measures and the practical use to which the combination of tests has since been put.

Measurement of the duration of the visual after-image, arising as the result of fixation of a rotating spiral, has been extensively used in the investigation of Eysenck's postulates (Holland (1960). It has been shown (Claridge, 1960) to discriminate hysterics and dysthymics, the latter having the longer after-effects; while it has been found to respond, in a manner consistent with this, to stimulant and depressant drugs (Eysenck *et al.* 1957; Costello 1960). Its relationship with sedation threshold can be seen in Fig. 2, the correlation here being 0·456 which, for 48 subjects, is significant beyond the 0·01 level of confidence.

Since that study was carried out, the combination of sedation threshold and Archimedes spiral after-effect has proved itself useful as a diagnostic tool, not only for discriminating the hysteric from the dysthymic, but also in the differential diagnosis of early schizophrenia and psychoneurosis. The latter has been made possible following the demonstration by Herrington and Claridge to appear that in young untreated schizophrenics the positive relationship between the two measures fails to appear. Instead, the relationship tends towards a negative one which, following treatment, is reversed and approximates more closely that found in the combined normal–neurotic group.

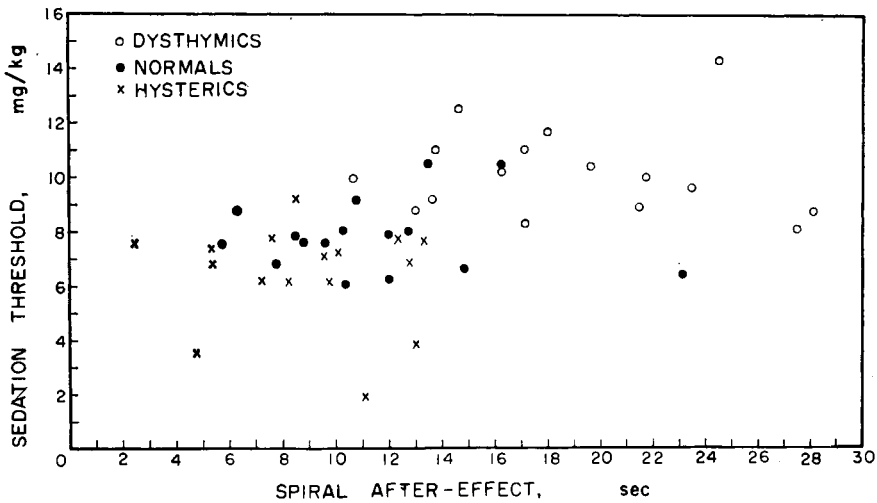


FIG. 2. Sedation threshold and spiral after-effect.

In investigating the relationship between personality and sedation threshold, two questionnaires were administered to each subject. These were the Taylor Manifest Anxiety Scale (MAS) and the Maudsley Personality Inventory (MPI) (Eysenck, 1959) which yields measures of extraversion (E-scale) and neuroticism (N-scale). On the total sample of 48 subjects, the sedation threshold correlated 0·355 ($p < 0·05$) with the N-scale and 0·383 ($p < 0·01$) with the MAS. With the E-scale the correlation of sedation threshold was not significant, "*r*" being −0·271. When the normal group alone was considered, however, this correlation rose to −0·524, which for 16 subjects is significant at the 0·05 level of confidence.

These findings tended to confirm, therefore, the hypothesis that the processes underlying sedation threshold are only partly associated with the behavioural dimension of extraversion and that when neurotic groups are included in the study of dysthymia–hysteria anxiety begins to play an increasingly important role. As a result of these findings on personality and performance, it was felt that we were not entirely incorrect in suggesting that there may be an interaction between arousal, as it relates to anxiety, and excitation–inhibition, as it relates to introversion–extraversion. It is now convenient to consider the possible nature of this interaction in more detail.

AUTONOMIC AROUSAL AND EXCITATION-INHIBITION

This discussion may profitably begin with the related concepts of anxiety, arousal, and drive. The notion that anxiety may act as a drive in facilitating learned connections is found in a number of papers, notably those of Taylor (1951), and Spence and Taylor (1951.) Although this particular

conception of anxiety has, perhaps, appeared most frequently in the context of Hullian or neo-Hullian analyses of behaviour, it would seem to be not essentially different from that inherent in the early ideas of Duffy (1934, 1941), when she suggested that emotion and motivation may be subsumed under the common heading of physiological arousal. Formulated in this way, the concept of arousal implies that emotion is an organized energizing process closely linked with the intensity aspect of drive. Although, apart from Duffy, other authors (e.g. Leeper, 1948; Webb, 1948) have at various times recognized this motivational aspect of emotion, the foremost contemporary adherent of an arousal theory of drive is Malmo (1958).

The most commonly used methods of measuring arousal level peripherally have been in terms of muscle tension, skin resistance or conductance, heart rate, blood pressure, and other somatic functions. The relationships of these measures of arousal to performance have been studied by many authors, including Duffy herself (1932), Freeman (1940), Malmo and Shagass (1952), Malmo and Davis (1956), Kling *et al.* (1959), Eason and White (1960), and Geldreich (1953); while, antedating all of these, Bills (1927), and Stroud (1931), among others, demonstrated that learning could be facilitated by artificially induced muscle tension.*

That there is a link between this conception of arousal and neurosis is clearly brought out in the writings of Duffy (1957) and more particularly of Malmo (1957; 1959), who has considered anxiety as a state of over-arousal. Of course, the idea is not new that there is a relationship between personality and autonomic activity, as factor analyses of physiological measures have shown. Of these, the most notable have been carried out by Wenger (1941; 1948), who reports a factor of autonomic imbalance which he considered differentiates normal and neurotic individuals. Similar factors of emotional lability and discharge control have been reported, respectively, by Theron (1948), and Freeman (1948), while, as we noted earlier, Eysenck considers his neuroticism dimension to be one of autonomic drive.

That the level of autonomic arousal is an important factor in neurotic reactions is, therefore, relatively well-established, although, as Eysenck has pointed out in a critical review (1960c) of factor analytical studies in this field, the exact relationships are by no means clear. One of the probable reasons for this is that little attention has been given to differences *within* neurotic groups on measures of autonomic activity. A notable exception to this, and one which is particularly relevant to our argument here, is the work of van der Merwe (1948). He compared groups of dysthymics, hysterics,

* Although we shall have occasion to refer to this point again, it should be noted here that while, as anticipated, the relationship between arousal level and efficiency of performance is usually a positive one, it is generally agreed that curvilinear, U-shaped functions appear when the whole range of arousal is studied, particularly with complex tasks. While Duffy, in her early papers, (*op. cit.*) seems to be the first "arousal theorist" to have noted this phenomenon, it did, in fact, have its first expression long before that in the Yerkes-Dodson law (Yerkes and Dodson 1908). Since then it has appeared in various contexts, notably in the work of Stauffacher (1937), Courts (1939), Vaughn and Diserens (1930), and Taylor and Spence (1952). The principle of the Yerkes-Dodson law has been incorporated into contemporary arousal theory by Hebb (1955), and Malmo (1958), while Yates (1960) has recently discussed its relevance to neurotic behaviour.

and normals on factor measures derived from the study by Theron *(op. cit.)* who used finger plethysmograph changes to establish factors of emotional lability and basic emotional tension. Van der Merwe found that the combined neurotic group of hysterics and dysthymics differed from the normal group in being significantly more labile, while the two neurotic groups taken separately did not differ significantly on this factor. On the other hand, on measures of basic emotional tension it was found that hysterics and dysthymics were ranged on either side of normal. Dysthymics deviated from normal in the direction of sympathetic predominance in their autonomic reaction, while hysterics deviated in the direction of parasympathetic predominance.

These findings would tend, then, to support our argument that dysthymics and hysterics differ in autonomic arousal, since upward changes in the level of arousal are assumed to involve predominantly an increase in the activity of the sympathetic division of the autonomic nervous system. In order to see how this hypothesis may be reconciled with Eysenck's excitation–inhibition theory of personality it is necessary to consider some of the recent evidence from neurophysiology.*

So far we have discussed arousal solely in terms of its peripheral accompaniments, since it was in the measurement of these functions that the concept had its origins in psychology. Its more recent popularity undoubtedly derives, however, from the rapid advances in knowledge about the ascending reticular formation. Since the demonstration by Moruzzi and Magoun (1949) that maintenance of the waking alert state is dependent on continuous activation from a reticular system in the brain stem, the concept of arousal in psychology has increasingly assumed the features of a central phenomenon. Reviews of work in this field of neurophysiology, and its implications for psychology, have been made by Lindsley (1956) and Samuel (1959), while its particular relevance to the problem of motivation has been discussed by Lindsley elsewhere (1957a), and by Hebb (1955), to whose neurophysiological theory of drive we have already referred.

Briefly, it may be stated that the activity of the reticular formation is known to depend on afferent input from the peripheral sensory receptors, and also on hormonal mechanisms, particularly of adrenal origin. The existence in the ascending reticular formation of an adrenaline-sensitive component at the mesencephalic level has led to the conclusion (Bonvallet *et al.*, 1954; Rothballer, 1956) that sympathetic tone is an important source of cortical and behavioural arousal. In summarizing this work, Dell, who elsewhere (1958) has demonstrated the importance of this system in physiological drives, concludes (1957): "In an animal at rest, an unexpected change in the outside world (producing the orientating reaction of Pavlov) or better, a painful stimulation, always produces an arousal effect and a sympathetic discharge (cardio-acceleration, adrenaline secretion etc.). The immediate primary nervous activation of the reticular systems is rapidly

* It must be stressed that physiological work is, of course, quoted only in so far as it relates to the argument here: the physiology of awareness and of drug action, even in so far as it is known, is more complex than the discussion here suggests.

intensified and subsequently maintained by a secondary humoral phase due to the circulating epinephrine. More generally it may be said that the level of the peripheral sympathetic tone is just as important a factor in maintaining the waking state and the level of alertness as the inflow of proprioceptive and exteroceptive stimuli... As a rule in the numerous situations analysed by Cannon (asphyxia, thermogenetic reactions, pain, fear, hunger, emergency states) which cause a sympathetic discharge, the reticular activating effect of epinephrine greatly enhances the central excitatory state."

This conclusion is of particular interest here because of the possibility, discussed above, that different neurotic types vary in their level of sympathetic tone. Apart from the work of van der Merwe (1948), the suggestion finds even more direct confirmation in the high activation (fast low voltage) EEG patterns associated with anxiety (Lindsley, 1951) and in the demonstration of raised plasma adrenaline levels in dysthymics (Weil-Malherbe, 1955).* Increased adreno-cortical activity, the secretion of both gluco-corticoids and mineralo–corticoids, has been demonstrated in anxiety states (Persky *et al.* 1956; 1959) and in anxiety in normals (Hoagland, 1960). This is of interest, since it is reported (Cook *et al.* 1960) that lack of gluco–corticoids increases sensitivity to barbiturates. Since it is known (King, 1956; Arduini and Arduini, 1954; French *et al.* 1953; Bradley and Key, 1958) that barbiturate anaesthesia initially and most markedly depresses the activity of the ascending reticular formation, one would expect from these varied clinical and experimental data that the sedation threshold would be raised in dysthymia.

Apart from work carried out on the reticular formation as an ascending activating system, other aspects of this field are relevant here. It is known, for example, that there are descending projections from various cortical areas to the reticular formation (French *et al.*, 1955; Kaada and Johannessen 1950). These have been found to be capable (Adey *et al.*, 1957; Hugelin and Bonvallet, 1957) of either facilitating or inhibiting ascending reticular activation, while cortical control of autonomic balance has been well documented (see French, 1957). There are means, therefore, in the nervous system whereby the cortex may influence its own arousal level, a factor which may be important in the changes which occur in hysterical suppression of anxiety. The accompanying conversion symptoms in hysteria are possibly also explicable along similar lines, since central control of arousal may occur at several levels in the nervous system, reticular stimulation having been shown (e.g. Galambos, 1956) to modify potentials at all stages along the sensory pathways, including at the receptor itself.

In view of the relationships found with peripheral measures of autonomic arousal, it is not surprising that the reticular formation should be considered to play an important role in psychological performance. Indeed, it has been shown (Fuster, 1957) that stimulation of the reticular formation in monkeys

* Incidentally here too a link can be discerned with the arousal theory of drive, since there is some preliminary evidence (Elmadjian, *et al.* 1956) of a positive relationship between level of pursuit rotor performance and the amount of excreted adrenaline.

improves both reaction time and the number of correct responses made on a visual discrimination task. Similar facilitating effects of reticular stimulation on perceptual discrimination in animals and reaction time in humans have been discussed by Lindsley (1957b), while Gastaut (1957), and Ingram (1957), respectively, have considered the role of the reticular formation in conditioning and learning.

At this point perhaps we may summarize some of the salient points arising from this discussion and present a modest neuropsychological interpretation of Eysenck's essentially molar concept of excitation–inhibition.

Despite the varied nature of the studies just reviewed, the links that can be discerned between them suggest a common thought underlying the different approaches. The idea that anxiety may act as a drive links up with attempts to measure the physiological accompaniments of emotion subsumed, with emotion, under the heading of "arousal". The usefulness of the latter concept is supported by neurophysiological evidence which has been able to characterize the central mechanisms determining arousal. These mechanisms have, furthermore, been shown to be influenced by similar autonomic changes which peripheralist arousal theorists have quoted as measures of arousal level. The next stage shows a common relationship with performance, of anxiety as a drive, of arousal as measured peripherally, and of neurophysiological activation. Finally, there is evidence that changes in these autonomically-linked arousal mechanisms may be an important determinant of the neurotic's behaviour.

This takes account of what we might call the "excitatory" aspects of behaviour. At the same time, the need to propose active inhibitory processes has been apparent for many years, in both psychology and physiology. The importance of these processes cannot be denied, if attention is paid to the tendency, found at all levels of behaviour, for equilibrium to be maintained by positive excitatory and negative opposing forces within the nervous system.

With this in mind, it seems to the authors that Eysenck's excitation–inhibition balance may be best conceived of, at a speculative neurophysiological level, as the result of an interaction between, on the one hand, subcortical and cortical processes and, on the other, between, at each of these levels, excitatory and inhibitory effects. In view of the evidence reviewed in this section, one of the major influences determining the excitation level must be the degree of arousal present. Since, apart from sensory maintained arousal, changes in arousal level may also occur in association with increased autonomic activity, positive shifts in the excitation–inhibition balance would seem to be a feasible outcome of the increased anxiety found in dysthymics. Comparable decreases in excitation level may be thought to occur in hysterics, perhaps as the result of a downward inhibitory discharge producing a damping of the arousal effect arising from autonomically-linked arousal mechanisms. This would appear to be associated with a predominance, in the hysteric, of parasympathetic activity in the autonomic nervous system.

This interpretation coincides closely with the original finding of Claridge (1960) that the differences between hysterics and dysthymics are partly explicable in terms of a factor of drive, which influences, at a central level,

the excitation–inhibition balance assumed to underly introversion–extraversion. Further implications for the theory of personality will be considered in a later section. In the meantime, however, it is necessary to describe a further experiment, the aims of which were to test a number of hypotheses arising from our analysis here.

SEDATION THRESHOLD AND AUTONOMIC AROUSAL

The analysis of dysthymia–hysteria presented in the previous section clearly suggests a number of hypotheses, the most important of which concerns the possibility of a relationship between sedation threshold and measures of autonomic arousal. If, as seems likely, we can regard the sedation threshold as a good index of the balance of excitation–inhibition in the nervous system and, if we are correct in assuming that this balance is partly determined, in neurotics, by the level of autonomic arousal, then a positive correlation would be expected between sedation threshold and a peripheral measure of autonomic activity.

It is interesting to note that other workers have considered this possibility. Shagass, for example, on the basis of his dual mechanism theory of anxiety mentioned earlier, proposed a similar hypothesis. However, he reports (1958) that an attempt, made with Sloane, to confirm it was negative. Using the Funkenstein test (Funkenstein *et al.* 1952) which it is claimed reflects the state of autonomic imbalance, they found no correlation between sedation threshold and blood pressure response to Mecholyl. Equally negative were the results of Ackner and Pampiglione (1958) who found no correlation between sedation threshold and an index derived from the finger plethysmograph. With regard to the latter study, however, it will be remembered that the authors report difficulty in defining sedation thresholds using an EEG technique.

Despite these negative results, it seemed worth carrying out a pilot study, at least, using a simple measure of autonomic arousal and the method of estimating sedation thresholds which, as we have already described, appeared to be more reliable than EEG methods. The measure of arousal chosen was that of heart rate, changes in which are a commonly recognized accompaniment of increased sympathetic activity during emotional excitement. It also has the advantage of being easily measurable in the form of a continuous record, a factor which was important for the experimental design to be used.

A second problem with which we were concerned was the relationship of our measures of excitation–inhibition and arousal to performance. The task chosen for this investigation was one of auditory vigilance, the particular test used having already been shown (Claridge, 1960) to differentiate dysthymics and hysterics. As expected, the latter showed poorer vigilance, due, it was proposed there, to their greater susceptibility to inhibitory processes under the monotonous conditions of a vigilance task. In view of the findings of the first experiment reported here, we could fairly confidently predict that sedation threshold would correlate significantly with performance on

this test. Of greater interest here, however, was the relationship between heart rate and performance. One of the main reasons for the choice of a vigilance task was that it allowed a measure of the heart rate to be taken during performance of the test. This procedure had the dual advantage (a) of presenting a standard stimulus situation to all subjects in order to compare their heart rates and (b) of providing a continuous and relatively lengthy sample of the heart rate, changes in which we would expect, from our hypothesis concerning arousal and excitation–inhibition, to be related to vigilance performance.

We have already, in the previous section, mentioned some of the studies which support the likelihood of a relationship between autonomic arousal and performance. One further study – by Bjerner (1950) – is worth considering here in a little more detail. Bjerner investigated the problem of involuntary rest pauses as indices of decrement in serial reaction time performance. He showed that a temporary slowing of the heart rate occurred during the "blocks" which appear after prolonged performance on the task. Also accompanying these blocks was the disappearance in the EEG of the normal *alpha* rhythym and the appearance of the large slow waves characteristic of sleep (see also Williams *et al.*, 1959).

The role played by rest pauses in perceptual–motor performance has been discussed from a number of viewpoints. They were considered early on by Bills (1931) to be a fatigue phenomenon, while Kimble (1949), followed by Eysenck (1956b), has re-analysed the problem in Hullian terms, putting forward the view that rest pauses form the basis of conditioned inhibition. Blocking has, however, been most extensively discussed by Broadbent (1958), who makes use of concepts from information rather than classical learning theory. In particular, he points to the similarity between serial reaction and vigilance tasks, while Ross *et al.* (1959) report some evidence of a relationship between arousal, as measured by skin conductance, and performance on the Mackworth clock-watching test. While, as in all true vigilance tasks, it was not possible in the present experiment to measure rest pauses directly, there seemed sufficient evidence to predict a relationship between vigilance decrement and heart rate.

We would tend, of course, to follow Eysenck, rather than Broadbent, in considering blocks to occur as the result of an active inhibitory process. In view of our earlier formulation of excitation–inhibition, it is of interest to consider briefly the possible course of events during a vigilance task of the type used here.

It may be said that the individual starts the test with a given level of alertness or drive which makes him more or less efficient at detecting the signals of the vigilance task. This condition of alertness reflects a state of central nervous excitability occurring in association with an increase in sympathetic activity, which is presumably influential cortically via the adrenergic component of the ascending reticular formation. The monotonous influx of stimuli must cause a state of affairs in the cortex and related structures such that a "damping down" of excitability occurs, a process which we may identify as that of inhibition. This damping effect must result not only in a reduction of cortical receptivity *per se*, but also, perhaps via descending

pathways, in a decrease in the facilitatory effects of autonomic arousal mechanisms.

One further problem was of interest, viz, the nature of the arousal concept itself. Inherent in the concept is the principle that arousal suggests a state of alertness, readiness, or drive, which is characteristic of the waking individual. It seemed likely, therefore, that any individual differences in autonomic arousal which we might find would be a function of the waking state, whereas during sleep such differences might disappear. It was decided, therefore, to take measures of the sleeping pulse, where it was expected that no differences between the groups would appear.

The aims of the investigation to be reported, therefore, may be summarized as follows:

(1) To investigate differences in the heart rate of dysthymics, normals, and hysterics when placed in a standard test situation.

(2) To determine the relationship between the level of autonomic arousal as measured by the heart rate and the excitation—inhibition balance as measured by the sedation threshold.

(3) To investigate the relationship between vigilance decrement and (a) sedation threshold and (b) heart rate.

(4) To determine whether there was any difference between the groups in sleeping pulse or whether any differences in autonomic arousal were a function solely of the waking state.

Selection and description of subjects

Untreated neurotic admissions between January and July 1960 were selected where the psychiatrist in charge of the case could make a definite diagnosis of anxiety state or hysteria. In each of these two neurotic groups there were 10 subjects, all of whom were male.

The normal control group consisted of 10 male volunteers who were engaged on various duties in the hospital, including that of nursing orderly, clerk, storeman, or laboratory technician.

The mean age and Matrices IQ for the three groups are shown, respectively, in Tables 2 and 3, where it can be seen that there is no difference between the groups on either variable.

TABLE 2

Mean age (in years) for each group

	Dysthymics	*Normals*	*Hysterics*
Mean	23·56	24·52	25·33
SD	2·728	5·389	5·872

F-ratio: 0·299, NS

TABLE 3

Mean Matrices IQ for each group

	Dysthymics	*Normals*	*Hysterics*
Mean	113·2	113·3	110·3
SD	7·86	9·23	10·95

F-ratio: 2·869, NS

During the testing session the Maudsley Personality Inventory (MPI) and the Taylor Manifest Anxiety Scale (MAS) were administered to each subject and the group means for each of these are shown in Tables 4 and 5 respectively.

TABLE 4

Mean MPI score for each group

	Dysthymics	*Normals*	*Hysterics*
	(a) E-scale score		
Mean	17·7	33·9	21·7
SD	11·38	5·84	10·97

F-ratio: 6·768, $p < 0·01$

t-tests (Dysthymics v. Hysterics: 0·872, NS
(Dysthymics v. Normals: 3·529, $p < 0·01$
(Hysterics v. Normals: 2·658, $p < 0·02$

	(b) N-scale score		
Mean	37·2	17·4	31·3
SD	10·87	7·77	10·02

F-ratio: 10·001, $p < 0·001$

t-tests (Dysthymics v. Hysterics: 1·300 NS
(Dysthymics v. Normals: 4·361, $p < 0·001$
(Hysterics v. Normals: 3·062, $p < 0·01$

TABLE 5

Mean MAS score for each group

	Dysthymics	*Normals*	*Hysterics*
Mean	18·5	5·1	14·4
SD	7·43	2·98	6·65

F-ratio: 11·744, $p < 0{\cdot}001$

t-tests: Dysthymics v. Hysterics: 1·449, NS; Dysthymics v. Normals: 4·735, $p < 0{\cdot}001$; Hysterics v. Normals: 3·286, $p < 0{\cdot}01$

It can be seen from these tables that the questionnaire scores of the three groups coincide closely with those of previous samples of neurotics tested. On extraversion (E-scale), hysterics fall midway between dysthymics and normals, while both neurotic groups are alike in having higher neuroticism (N-scale) scores and manifest anxiety scores than normals.

*Experimental procedure**

A sedation threshold was determined for all subjects according to the technique described earlier. This, it will be recalled, gives a threshold in terms of milligrammes of sodium amytal received per kilogramme of body weight.

The vigilance task consisted of a 30 min tape-recording of random digits read out at the rate of one per sec. Interspersed in the series were a number of "signals" which consisted of three consecutive odd digits. The signals occurred with a frequency of one per min, the exact position of each signal within each minute being determined randomly. The subject was asked to try and detect the signals and when he did so, to respond by tapping a morse key. Two scores were taken from the test, the total number of signals detected, the maximum being 30, and the number detected within each 5 min period of the test.

During performance of the vigilance test, the subject lay on a bed while a continuous record of the heart rate was made on a standard Cossor electro-cardiograph, electrodes being attached to the right arm and left leg of the subject. A 30 min sample of the heart rate was, therefore, obtained, the beats being recorded on a moving paper roll and afterwards counted. For scoring purposes, the record was divided into six 5 min periods, coinciding with the 5 min periods of the vigilance task. For each 5 min period, the total number of heart beats was counted and this total divided by 5 to give an average minute pulse rate for that period. Six of these measures were, of course, obtained from each subject.

* To take care of diurnal variations in response, sedation thresholds were always measured between 10 and 12 a.m. and waking heart rate between 3 and 4.30 p.m.

The final measure taken was that of the sleeping pulse, of which one minute samples were taken on 3 consecutive nights at 2 a.m., 3 a.m, and 4 a.m., the nine measures thus obtained being averaged to give a single measure of the sleeping pulse. While it was possible to get measures of the sleeping pulse for all the patients, such was not the case for the normal group, some of whom lived outside the hospital, while others had left before it was decided to take this measure. It was decided therefore, for comparative purposes, to obtain sleeping pulses, using the method outlined, of 20 normal control subjects of similar age and background as those used in the original control group.

Results

(a) *Group differences.* Turning first to the sedation threshold, it can be seen from Table 6 that there is, as expected, an overall significant difference between the three groups, dysthymics having the highest and hysterics the lowest mean threshold, with normals intermediate. The results differ from those reported earlier in respect of the difference between dysthymics and normals, which, in this sample, is not significant. This is due to the much lower mean for this group than for the previous sample of dysthymics tested.

TABLE 6

Mean sedation threshold (in mg/kg) for each group

	Dysthymics	*Normals*	*Hysterics*
Mean	8·80	8·34	6·23
SD	1·138	1·871	1·670

F-ratio: 6·625, $p < 0·01$

t-tests (Dysthymics v. Hysterics: 3·422, $p < 0·01$
(Dysthymics v. Normals: 0·614, NS
(Hysterics v. Normals: 2·810, $p < 0·01$

The group differences in heart rate can be most clearly seen graphically, as in Fig. 3, where the average pulse rate for each 5 min. period has been plotted. It can be seen from this figure that the three groups are completely separated at all stages, dysthymics showing the highest and hysterics the lowest heart rate, with normals intermediate. In order to analyse these trends statistically, linear regression lines were fitted to each subject's scores, the data being taken in the form shown in Fig. 3. This technique gives two regression coefficients, viz. "*b*" representing the starting level and "*a*" representing the average change per trial (in this case average rate of decline over six 5 min. periods). In Table 7 are shown the group means for each of these measures, together with a summary of the statistical analysis. It can be seen that the coefficient "*b*" which is the best single measure

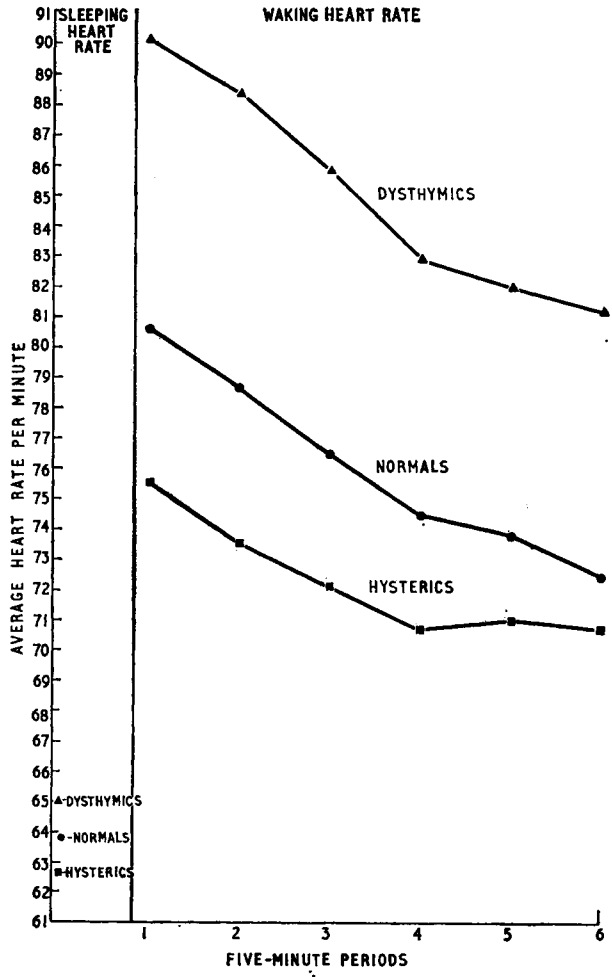


FIG. 3. Mean sleeping pulse and mean change in waking pulse during vigilance performance.

of heart rate, discriminates the three groups at an acceptable level of confidence, although this is mainly due to a significant difference between hysterics and dysthymics, the other group comparisons being non-significant.

The group means for the coefficient "*a*" reflect the trends shown in Fig. 3, dysthymics showing somewhat greater decline in pulse rate than hysterics and normals. This seems to be due to the tendency, not uncommon in this kind of analysis, for the amount of change in a function to be related to its basal level, a tendency which is supported in this instance

TABLE 7

Waking heart rate: mean score on two measures for each group

	Dysthymics	*Normals*	*Hysterics*
	(*a*) *Coefficient "b" (Initial level)*		
Mean	91·75	81·83	75·49
SD	11·759	14·075	11·886

F-ratio: 3·797, $p < 0·05$

t-tests (Dysthymics v. Hysterics: 2·733 $p < 0·02$
(Dysthymics v. Normals: 1·667, NS
(Hysterics v. Normals: 1·066, NS

	(*b*) *Coefficient "a" (Rate of decline)*		
Mean	−1·91	−1·63	−0·94
SD	1·211	0·838	1·136

F-ratio: 1·945, NS

by a significant correlation between the two regression coefficients ("*r*" = − 0·53, $p < 0·01$). This relationship possibly arises because the increase in arousal induced at the beginning of the vigilance task is greater in the dysthymics than in the other two groups. They would, therefore, have to decline further to reach their "natural" waking arousal level, which we might surmise is represented by the heart rate obtaining at the end of the task. In this respect, it is interesting to note that even at this stage the three groups are still well separated.

This point leads us naturally to a consideration of the sleeping pulse measure, the group means for which are shown in Table 8, where, it should

TABLE 8

Mean sleeping heart rate for three groups

	Dysthymics	*Normals**	*Hysterics*
Mean	64·95	63·60	62·73
SD	4·513	4·330	6·282

F-ratio: 0·479, NS

* — This mean is for 20 subjects not taking part in the main experiment.

be recalled, the normal group is a separate sample of 20 subjects. It is clear that there is no difference between dysthymics, hysterics, and normals in sleeping pulse, the group means being virtually identical. This seems to be an important finding, since it indicates that only during the waking state do individuals differ in their level of autonomic arousal, the differences disappearing during sleep.

The extent to which differential arousal occurs as a result of being in a waking, alert state was estimated for the two neurotic groups only by taking a measure based on the difference between sleeping pulse and average pulse rate during the first 5 min of vigilance. Dysthymics showed an average increase over their sleeping level of 25·19 beats per min, S.D. 13·345, while hysterics showed an increase of only 12·75, S.D. 8·892. The difference between these two means was significant at the 0·001 level of confidence, "t" being 7·924.

Finally, we may turn to the group differences in vigilance performance. These are shown graphically in Fig. 4, where are plotted the average number of signals detected by each group in each 5 min period of the test. It can

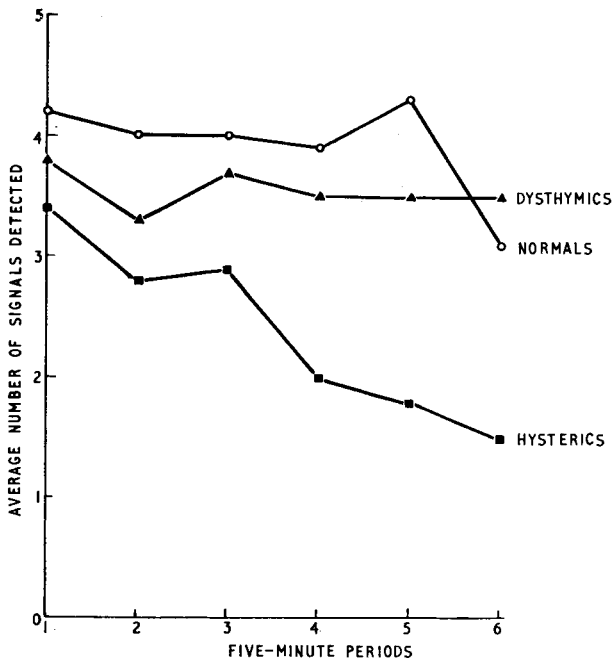


FIG. 4. Vigilance task performance of normal, dysthymic and hysteric subjects.

be seen that hysterics show poorer vigilance than dysthymics at all stages of the test, but, not confirming previous work with this task, normals are superior to both neurotic groups. This tendency for normals to fall in an intermediate position on both sedation threshold and heart rate, yet to

show superior vigilance, is of some significance, but a discussion of the point will be delayed until the next section.

In Table 9 are shown the group means for the total vigilance score and for the "*a*" coefficient of regression which was calculated on each subject's data in order to give a measure of rate of fall-off in vigilance (the "*b*" coefficient is of little interest and will not be considered here). The tendency, shown in Fig. 4, for hysterics to decline more in vigilance

TABLE 9

Vigilance test: mean score on two measures for each group

	Dysthymics	*Normals*	*Hysterics*
	(*a*) *total vigilance score*		
Mean	21·3	23·5	14·4
SD	7·96	4·01	7·70

F-ratio: 4·388, $p < 0·05$

t-tests (Dysthymics v. Hysterics: 2·153, $p < 0·05$
(Dysthymics v. Normals: 0·686, NS
(Hysterics v. Normals: 2·839, $p < 0·01$

	(*b*) *Coefficient "a" (Rate of fall-off)*		
Mean	−0·02	−0·09	−0·39
SD	0·147	0·468	0·295

F-ratio: 3·191, just fails significance.

than the other groups is reflected in the group means for the "*a*" coefficient, but on analysis of variance, the differences between the groups just failed to reach significance at an acceptable level of confidence. In terms of total vigilance score, however, there was an overall significant difference between the groups and (see Table 9) "*t*" tests indicated that hysterics had a significantly lower vigilance score than the other two groups. Normals and dysthymics were not, however, significantly different.

(b) *Intercorrelations between measures.* The first correlation calculated over the total group of 30 subjects was that between sedation threshold and a measure of heart rate in terms of the "*b*" regression coefficient which, it will be remembered, reflects that level obtaining at the beginning of the vigilance task. The value for "*r*" was found to be 0·39, which is significant at the

0·05 level of confidence. This suggests, then, that individual differences in tolerance to barbiturates are related to differences in the level of autonomic arousal, thus supporting the view developed in this paper. A comment should be made, however, about individual variations in this respect, variations which tend to be masked by the overall correlation. By taking only a single measure of arousal we did, of course, ignore the factor of autonomic response specificity which has been emphasized by Lacey and his colleagues (Lacey, 1950; Lacey and Lacey, 1958; Lacey *et al.*, 1953). This factor, which may be described as the tendency for individuals to show idiosyncratic patterns of autonomic response, operated in the present sample in a number of ways.

For example, one subject, who undoubtedly fell into the hysteric group and had a very low sedation threshold of 3·82 mg/kg, showed an average pulse rate at the beginning of the vigilance task of 93·2 per min. The particular symptom shown by this patient during stress was blackouts following hyperventilation, a reaction which induces peripheral vasodilatation and, therefore, an increase in the heart rate. At the other extreme was a dysthymic who showed an average pulse rate lower than the mean for the hysteric group. This man was known to show an enthusiasm for athletics which was above average, a state which tends to lower the basal heart rate.

This specificity of response partly accounts, of course, for the relatively low correlation between heart rate and sedation threshold. It is, however, encouraging that a significant correlation should have appeared despite this fact and, far from negating the results, it suggests the need for a more thorough investigation of the problem using a wider variety of autonomic measures.

The next series of correlations calculated were between sedation threshold and heart rate and the two measures taken from the vigilance task. Total vigilance score correlated with sedation threshold to the extent of 0·57, which is easily significant at the 0·01 level. With the heart rate "*b*" coefficient total vigilance correlated only to the extent of 0·24, however, this being non-significant. On the other hand, heart rate correlated significantly with rate of fall-off in vigilance ("*r*" equals 0·41, $p < 0·05$)*, while sedation threshold just failed to do so ("*r*" equals 0·35). To determine whether there was any correlation between decline in vigilance and decline in pulse rate, the two "*a*" coefficients for these variables were correlated. The resulting value for "*r*" was, however, only −0·13 which, apart from being non-significant, is in any case in the wrong direction.

This latter correlation arises mainly because dysthymics show the least decline in vigilance, yet the greatest decline in pulse rate. It is of interest, however, because it may relate to the finding noted earlier, that normals are in terms of total score, superior to dysthymics on vigilance but lower on heart rate. It is possible that the relationship between vigilance and autonomic arousal is basically curvilinear, a phenomenon which, as we

* The reader is reminded that decline in vigilance was scored in terms of negative values of the "*a*" regression coefficient. This positive correlation should, therefore, be interpreted as meaning that vigilance decline is greatest where the basal heart rate is low.

discussed in a previous section, is not unusual in this field. Reference to Figs. 2 and 3 may help to clarify this suggestion. It seems likely that at the beginning of the vigilance task, the arousal level of dysthymics was beyond the optimum for efficient performance. As arousal level declined (necessarily more rapidly than in other groups because of their higher starting point) performance efficiency eventually reached its maximum and so overall decline in vigilance was somewhat less than in normals who, while at optimum arousal level at the beginning, were, by the end of the task, falling below optimum as arousal fell. Hysterics, of course, were below optimum from the very beginning and fell even further below as the test proceeded.

Attempts to isolate this curvilinear relationship were unfortunately unsuccessful, partly because of the small range of scores forthcoming from the vigilance test. The above interpretation must, therefore, remain *post hoc*. Nevertheless, the relationship seems a likely one in view of the frequency with which other workers in the field have reported it. On the whole, the correlations in this part of the study must remain suggestive rather than definitive, although one conclusion can be drawn with some certainty, namely, that autonomic arousal and the mechanisms mediating barbiturate tolerance have a common relationship with the processes underlying attention.

The final relationships considered were those with the questionnaire measures. These were all insignificant, the highest correlation found being that between heart rate and the E-scale of the MPI, which had a value of −0·28. This was not significant for 30 cases, but suggested a slight tendency for autonomic arousal to be higher in introverts than extraverts. With the N-scale and the MAS, heart rate correlated only to the extent of −0·01 and −0·09. The correlations of sedation threshold with the N-scale, the E-scale, and the MAS were, respectively, −0·11, 0·10, and −0·11. These latter results do not, therefore, confirm those of the previous study of sedation threshold and clearly throw no further light on the relationships with personality as reflected in questionnaire responses.

FINAL DISCUSSION

The results of the investigation just reported seem to be important in two respects. First, it has been possible to define some of the physiological correlates of the essentially molar concept of excitation–inhibition. Secondly, a link has been made between two current theories of behaviour. The first is that theory—arousal theory—which analyses performance in terms of excitatory variables, reflected physiologically in the activity of the autonomic nervous system. The second theory is that of Eysenck's school of psychology, which considers changes in performance to be due to inhibitory processes, which, while having no defined physiological identity, have usually been assumed by workers in that school to be essentially cortical in origin. Autonomic activity has mainly been considered in the latter theory to be relevant to the neuroticism dimension of personality, rather than to introversion–extraversion.

There seems little doubt that differences in the level of autonomic arousal are an important factor determining the respective performance levels of dysthymics and hysterics. However, in our theoretical analysis, in a previous section, of the excitation–inhibition concept, we proposed the view that the autonomic changes occurring in neurosis were only one influence affecting the central excitation–inhibition balance. We suggested that these changes played a prominent part only in neurotic groups, where the level of autonomic lability tended to be high and to result in the physiological changes associated with anxiety. By proposing this additional influence on the excitation–inhibition balance, it was felt possible to account for the apparent lack of identity between excitation–inhibition and the behavioural dimension of introversion–extraversion when neurotic groups were being studied. In view of this modification to Eysenck's theory, it is of interest to consider further the possible relationships between, on the one hand, autonomic arousal and excitation–inhibition and, on the other, between each of these concepts and personality, particularly with regard to introversion–extraversion as an explanatory concept for dysthymia–hysteria.

Several possibilities suggest themselves, the first that must be considered being that the excitation–inhibition balance of Eysenck is explicable physiologically entirely in terms of autonomic functioning. For example, it may be the case that the underlying physiological concomitant of introversion–extraversion is a continuum running from sympathetic to parasympathetic predominance. Neuroticism would then consist simply of the tendency to "swing" autonomically, dysthymics towards greater sympathetic and hysterics greater parasympathetic predominance. This model could account for the shift in excitation–inhibition initially proposed to account for the disproportionate excitatory and inhibitory effects seen in neurotics. It also finds some parallel in Shagass's dual mechanism concept of anxiety referred to at the beginning of this paper. Presumably, according to Shagass, the dysthymic feels anxiety in terms of increased sympathetic activity, while the hysteric feels anxiety mediated through parasympathetic mechanisms. Since it is adrenergic mechanisms which are relevant to the sedation threshold, it will be the neurotic introvert who has the highest and the neurotic extravert who has the lowest threshold.

This analysis of introversion–extraversion solely in terms of autonomic activity is a plausible one which Eysenck (1960) himself has considered. After reviewing the evidence, however, and quoting particularly the work of Wenger referred to earlier, he concludes that the evidence for such a hypothesis is contradictory, a fact which, in such a precise field as this, must be considered particularly damaging.

While considering this possibility, however, mention should be made of the extensive analysis of personality by Cattell (1957), parts of which bear on this problem. Cattell reports a source trait which he names "Parmiathrectia". This refers, at the positive end, to a state of parasympathetic predominance in autonomic function and shows a positive loading on the factor of extraversion–introversion. There are, however, two other findings reported by Cattell which militate against a simple interpreta-

tion of his data. First, he reports a *positive* correlation between parmia and another source trait of "thinking introversion". Secondly, from his analysis of autonomic measures he concludes that sympathetic predominance is not the negative end of a bipolar factor of parasympathetic–sympathetic activity. It is, instead, a separate factor which he terms "adrenergic" and which, curiously, again is *positively* loaded on extraversion. These findings are too inconsistent to be regarded as critical to the argument relating introversion–extraversion solely to autonomic activity. Apart from that, however, there are other reasons for rejecting this simple model of personality.

One major reason is that it fails to take account of the undoubted importance of other types of nervous activity upon which individuals almost certainly differ. We have discussed some of these in considering the possible neurophysiological basis of molar excitation–inhibition and have quoted particularly the need to propose, as Eysenck, following Pavlov, has done, a cortical inhibitory process which differentiates individuals of various personality types. While the analogous homeostatic purpose of the parasympathetic system at the autonomic level is most well-known, it would be rash to ignore the restraining role of inhibitory processes at other levels of nervous function. Perhaps we may comment briefly on the reasons, culled from our own work, for regarding cortical inhibition as a functionally separate process which is related to personality.

The first point arises from the second experiment reported here. There, all subjects were administered the Archimedes spiral, the after-image from which is thought to reflect the degree of cortical satiation (inhibition) induced by the rotating spiral. Despite the significant positive correlation between sedation threshold and spiral after-effect and between sedation threshold and heart rate, the correlation between heart rate and spiral after-effect was not significant, "*r*" being only 0·19. This suggests that the common relationship between sedation threshold and spiral after-effect does not arise principally from autonomic influences. The pattern of correlations rather suggests that the major part of the sedation threshold measure is determined by the level of autonomic arousal, while cortical satiation is mainly responsible for the spiral after-effect. The correlation between the two and the positive sign of that between spiral after-effect and heart rate may arise partly because of overlapping variance and partly because the two processes may, in fact, interact in neurotics.* How this might occur will be developed later.

More critical evidence bearing on this problem comes from some work, already mentioned briefly, on the relationship between sedation threshold and spiral after-effect in psychotics (Herrington and Claridge, *op. cit.*). There, it may be recalled, there was a negative correlation between spiral after-effect and the sedation threshold, rather than the positive one usually

* Since writing this paper, further evidence has emerged which confirms this suggestion that the spiral after-effect and sedation threshold measure processes which, while they may interact, are fundamentally different. In a study, as yet unpublished, it has been found that the sedation threshold correlates positively and significantly with indices of blood pressure response on both the Funkenstein and cold pressor tests. Each of these autonomic measures has, however, virtually zero correlation with the spiral after-effect.

found with neurotics and normals. A possible interpretation of this, bearing in mind the first finding described above, is that, in psychotics, a dissociation occurs between sensory determined excitation–inhibition and autonomic arousal. Such a possibility could provide a framework for understanding schizophrenic symptoms such as, on the one hand, flattening of affect and retardation and, on the other, inappropriateness of emotional response and ideas of reference.* If psychotic illness does indeed represent a state of affairs where autonomic arousal and cortical inhibition are naturally separated, then this presents an exciting prospect for future research in this important area.

We must now turn to the second objection to a purely autonomic explanation of dysthymia–hysteria. This concerns the nature of the neurotic reaction itself. It seems, to the authors, an unlikely hypothesis that there are, in fact, two kinds of anxiety. It is true, of course, that individuals differ in the pattern of autonomic response they show under stress, as we have already briefly indicated in quoting the work of Lacey *(op. cit.)*. On the other hand, there is no *a priori* reason for thinking that these patterns of reaction are related to the broad division of personality into dysthymic and hysteric. It seems more likely that all neurotic individuals show excessive lability in their autonomic reaction to stress, the patterning being specific and unrelated directly to dysthymia–hysteria. This excessive lability will tend, in all individuals, to be reflected predominantly in increased sympathetic activity, simply because of the physiological nature of this division of the autonomic nervous system, i.e. as serving an emergency function. On the other hand, as Lacey has pointed out so lucidly (1956), the greater the sympathetic response, the disproportionately greater will be the restraining homeostatic effect of the parasympathetic reaction reflexly initiated. In some individuals, therefore, an excessive parasympathetic response may occur, perhaps as an "overshoot" effect of previous sympathetic stimulation. In the clinical setting, of course, this is well known. Such clear parasympathetic reactions as early morning diarrhoea and frequent micturition appear, if anything, more commonly in the dysthymic than in the hysteric.** If this analysis is correct, how do we account for the suggestions such as that of Van der Merwe (*op.cit.*) that dysthymics and hysterics are characterized by, respectively, sympathetic and parasympathetic predominance? A possible explanation forms the basis of a theoretical model of dysthymia–hysteria alternative to that discussed above.

It is suggested, as Eysenck has done, that individuals differ in the ease with which inhibitory processes are called into play, variations in this respect being reflected, in the non-neurotic population, in the behavioural

* In this respect it is tempting to find a link with the analysis of psychosis by Payne (Payne and Hewlett, 1960) who has demonstrated factors of retardation and over-inclusiveness of thinking in psychotics. Retardation may occur in patients where autonomic arousal is lowered in association with excessive cortical inhibition, while over-inclusiveness, and associated paranoid ideas, may arise as a result of a severe reduction in the normal cortical inhibitory process.

** The type of autonomic discharge may, in fact, depend very much on variables other than personality, particularly the precipitating situation. Gellhorn (1953), for example, has carefully analysed these various factors, utilizing experimental and clinical evidence.

dimension of introversion–extraversion. Two kinds of stimulation may be said to elicit inhibition: afferent stimulation arising from the sense organs, and stimulation occuring in association with excessive lability of the autonomic nervous system (i.e. autonomic arousal). In the non-neurotic population, it is inhibition arising mainly from afferent stimulation which has usually been studied, e.g. in terms of sensory after-images. In this group, correlations should therefore appear between such measures of inhibition and extraversion. Of course, under these conditions, there may be differences between normal introverts and extraverts in their prevailing *level* of sympathetic arousal, because any sensory stimulus will produce a sympathetic alerting response. This response should be more persistent in the introvert because of his slower adaptation to sensory stimulation.

However, in neurotic groups an additional factor will be present, viz. that of autonomic *lability* or the tendency to excessive change in the level of autonomic response. This, within the limits of response specificity, will be similar for all neurotics. The *internally-aroused* stimulation will, also, as with afferent stimulation, call into play inhibitory processes. Because of their basically extraverted (easily inhibited) tendencies, hysterics will tend to "damp down" or inhibit this increase in autonomic stimulation, which would be potentially felt as anxiety. This damping down process will tend to reduce autonomic arousal to an abnormally low level so that it no longer becomes, as in the normal group, a relatively small effect but one having a real influence on cortical receptivity. This mechanism may be thought of as serving two functions. First, it will tend to reduce the level of autonomic arousal directly and, because of the predominantly (although not exclusively) sympathetic nature of anxiety, it will tend to result, in the hysteric, in an apparent predominance of parasympathetic activity, which is perhaps better termed "non-sympathetic." Secondly, an actual reduction of afferent stimulation, in the form of conversion symptoms such as blindness or paralysis, will tend to remove the environmental source of the anxiety.

The excessive autonomic response of the dysthymic results in a simpler picture. Inhibition of the internal stimulation will be less readily elicited and it will be felt as "free-floating" anxiety, occurring in association with somatic symptoms. There will be a tendency for this heightened arousal to increase cortical receptivity above the level of the normal introvert and, within the limits of the U-shaped functions already discussed, to facilitate learned connections.* The consideration given here to the two sources of

* The tendency for damping down of autonomic activity to occur under stress may, perhaps, be thought of in terms of a threshold of response. For example, it is not uncommon in the writers' experience to find apparently dysthymic personalities (having high sedation thresholds etc.) who show what appear to be clear hysterical symptoms, mainly in the form of amnesia following intense stress. Looked at another way, even the non-neurotic individual may show similar reactions if there is sufficiently severe stress, as in wartime. These possibilities illustrate the difficulty of selecting neurotic groups solely on the basis of symptomatology. In this respect, it is interesting to note the work of Foulds (Foulds, 1959; Foulds and Caine, 1958), who, using two groups of tests, was able to differentiate neurotics, first in terms of the underlying personality, whether introvert or extravert, and secondly in terms of the type of neurotic breakdown, whether hysteric or dysthymic.

stimulation which might give rise to inhibition coincides well with emphasis laid by Dell (1957) on the "internal milieu" as a means, additional to afferent stimuli, of maintaining cortical excitation. This aspect of the analysis will shortly be developed in a little more detail, but first of all it is necessary to consider one other point.

So far we have assumed that the additional influence of autonomic arousal on the E–I balance is operative solely during the neurotic breakdown itself. This is to say, we have presented a simple model of personality where the lack of correspondence, in neurotics, between the state of E–I balance and the questionnaire measure of extraversion arises because of a *temporary* shift due to increased or decreased autonomic response. According to this interpretation, it would be only during the neurotic breakdown that the E–I balance, as reflected in objective tests, failed to coincide with the degree of extraversion, as measured by the questionnaire. There is some evidence that this might be true. Shagass *et al.*, for example, have demonstrated (1957) decreases in the sedation thresholds of some dysthymics when their anxiety symptoms are relieved. Similar changes in the opposite direction in hysterics do not appear to have been reported, but these are a possibility. However, although it seems probable that such temporary shifts in the E–I balance may contribute partly to the differences between neurotics and their normal counterparts, it seems unlikely that they account entirely for the lack of correspondence between extraversion and excitation–inhibition.* It seems more likely that the inherent tendency of all neurotics to be autonomically labile itself influences the development of introverted tendencies and, therefore, the questionnaire response. It is of interest to consider how this might occur.

The possibility to be considered makes use of one of the explanations given by Eysenck and Claridge (1962) for the E and N scales of the MPI to correlate significantly when neurotic groups are included in the correlation ("r_{EN}" averages about $-0{\cdot}45$). The explanation given for this is based on the recognition by Eysenck (1956a) of two types of social shyness. He considers that the normal introvert is unsociable because he does not care to mix with people, although he is willing to do so if necessary. The neurotic introvert, in the other hand, is unsociable because he is afraid of other people, even though, in fact, he would like to mix with them.

It is of interest to link this distinction with the two kinds of stimuli which we have suggested help to maintain the E–I balance. It is possible that individual differences in normal introversion arise as a result of differences in the degree to which afferent stimulation is necessary to maintain an adequate level of cortical excitation. The normal extravert, therefore, would, because of rapid habituation, tend to require continually changing afferent

* A quite separate point, of course, is that the inherent tendency of neurotics to respond in particular ways to stress will probably eventually result in relatively permanent changes in their physiological status. Malmo (1957), for example, has put forward the view that prolonged overarousal, due to chronic anxiety, will produce weakening of cortical inhibitory processes. This, and allied hypotheses, has also recently been discussed by Tong and Murphy (1960).

input. One aspect of this would be his tendency to seek the stimulation of other people. The excitation level of the normal introvert, on the other hand, would be maintained by less frequently changing and lower levels of afferent stimulation. It would tend to be self-maintained for longer periods, a capacity which would be reflected in a decreased need for the stimulation of social intercourse.

We should, perhaps, re-emphasize here a point made earlier, viz. that the greater persistence of stimulation in the introvert may result in a higher prevailing level of sympathetic arousal because of the tendency for any sensory stimulation to result in a sympathetic arousal response. This is, of course, a different aspect of nervous function from the excessive *lability* of autonomic response, which is essentially neurotic.

Neurotic introversion may, in fact, arise in an entirely different way. If all neurotics, irrespective of potential neurotic type, are highly reactive autonomically, then there will be a tendency for them, in childhood, to show increased fear responses to other people, i.e. to become socially shy. Because of the large part played by questions on sociability in the E-scale of the MPI, this introverting tendency will appear to have a biasing effect on the extraversion dimension, along which the neurotics will in fact be distributed independently of their neurotic tendencies. While in the case of the potential dysthymic, this bias will simply exaggerate the degree of introversion already existing, the potential hysteric will be characterized by a lower level of behavioural extraversion than his constitution would suggest. It is easy to see, therefore, how the hysteric may emerge with only moderate scores on questionnaire measures of extraversion and how correlations with measures of neuroticism may appear.

This analysis of extraversion has some implications for the effects of experimentally induced sensory deprivation. On the basis of the theory presented here, it would be predicted that the normal introvert would tolerate sensory deprivation better than the normal extravert. Evidence that this is so has been presented by Petrie *et al.* (1958; 1960) who found differences in the MPI E-scale scores of good and poor tolerators of reduced sensory input. The reverse relationship was found by the same authors between the E-scale and the tolerance for pain, which, in contrast to sensory deprivation, they regarded as positively related to satiability. While considering this topic, however, mention should also be made of evidence (Smith and Lewty, 1959) of an inverse relationship between neuroticism and the tolerance of sensory deprivation. In the study quoted, which employed normal volunteers, there was a distinct tendency for the more neurotic in the group to emerge earlier from the deprivation room. A product moment correlation calculated by the present authors on the data of that study revealed a value for "*r*" of $-0{\cdot}84$ ($p < 0{\cdot}001$) between hours of deprivation and score on the MMQ. Quite apart from any relationship with extraversion, then, neurotic traits would also appear to play an important role in determining an individual's response to sensory deprivation, perhaps because extreme isolation may itself be a form of stress for the neurotic comparable to its opposite, excessive sensory stimulation. It would, of course, be of interest to compare, on the same group, the respective correlations between hours

of deprivation and scores on the E- and N-scales of the MPI. Attempts are at present being made to do this, using the original sample of Smith and Lewty.

SUMMARY AND CONCLUSIONS

In this paper, an attempt has been made to present, in an historical fashion, the development of a theoretical analysis of introversion–extraversion and dysthymia–hysteria. The starting-point for this analysis was an experimental investigation of Eysenck's hypothesis that the performance differences, on objective tests, between hysterics and dysthymics arise because of variations in the degree of extraversion present in these groups. While a factor analysis indicated that this was partly true, an additional factor present suggested that hysterics and dysthymics also differ in drive level. This finding was linked to the clinical fact that these neurotic types differ in their level of anxiety, the hysteric being characterized by so-called "*belle indifference*". The hypothesis proposed at that point was that increases and decreases in anxiety level in neurotics caused a shift in the excitation–inhibition balance (proposed by Eysenck to underlie introversion–extraversion), so that the state of E–I balance in dysthymics and hysterics was no longer entirely predictable from their respective degrees of extraversion.

In order to provide further evidence for this viewpoint, and to obtain an independent measure of the excitation–inhibition balance, a new method of measuring the sedation threshold was devised. This method, which proved more reliable than EEG methods, made use of the effect of intravenous Sodium Amytal on a simple test of attention. Application of this technique to groups of normals, hysterics, and dysthymics indicated that, supporting previous work, the three groups were significantly differentiated, with dysthymics and hysterics occupying, respectively, the upper and lower extremes. Correlations were found between sedation threshold and measures from other objective performance tests, while the pattern of correlations with measures of extraversion, anxiety and neuroticism supported the hypothesis that the differences between hysterics and dysthymics could not be explained entirely in terms of extraversion.

Consideration was then given to the way in which differences in drive may influence the excitation–inhibition balance in neurotics. The analysis began with a review of some aspects of arousal theory. The first point considered was the relationship between autonomic measures of arousal and performance. This was linked with (a) the relationship between anxiety and autonomic lability and (b) the tendency reported for hysterics and dysthymics to differ on measures of autonomic arousal. The psychological concept of arousal was then related to recent work on the reticular formation as the central neurophysiological source of cortical activation. Consideration was given particularly to the suggestion that the level of sympathetic tone may be a second major source, additional to afferent input, of cortical excitation. Upward shifts in the excitation–inhibition balance of dysthymics seemed, therefore, a feasible outcome of the heightened anxiety occurring in these patients. The possibility of downward inhibitory effects were thought

to account for the conversion of anxiety in hysterics and for the downward shift in the E–I balance of these patients.

A second experiment, not previously reported, was then described. Here the aim was to relate the sedation threshold to a measure of autonomic arousal and each of these, to performance on a vigilance task. During performance of the latter, a continuous record was taken of the heart rate of groups of normals, dysthymics and hysterics. Intercorrelations were found between heart rate, sedation threshold and vigilance decrement, while group comparisons indicated that dysthymics were highest and hysterics lowest on sedation threshold and waking heart rate. It was noted, however, that the differences in heart rate were a function solely of the waking state, normals, hysterics and dysthymics having virtually the same mean on a measure of the sleeping pulse.

In the final discussion of results, it was suggested that there was considerable support for the thesis that the excitation–inhibition balance of hysterics and dysthymics is influenced by changes in the level of autonomic arousal, even though variations in the level of inhibition in non-neurotic groups may be uninfluenced by this factor. While it was felt that there was some evidence that the shifts in the E–I balance were temporary ones due to the neurotic breakdown, the possibility was considered that they were a more permanent feature of the neurotic personality. In considering this point, use was made of the distinction between "normal" and "neurotic" introversion. The first, it was suggested, may arise through individual variations in the degree to which afferent input was necessary to maintain an adequate level of excitation. The second type of introversion was thought to develop as a result of conditioned fear responses, in all neurotics, to other people. This "introverting" effect of neuroticism would account partly for the tendency of hysterics to have only moderate scores on measures of extraversion and for the latter to correlate significantly with measures of neuroticism.

This extension of Eysenck's theoretical framework has a number of advantages over the original hypothesis that dysthymics represent *solely* the extremes of a unitary trait of introversion–extraversion. Not the least advantage is that means have been suggested whereby the cortical E–I balance, hitherto considered only as the process underlying introversion–extraversion, may be influenced by autonomic arousal mechanisms, measures of which have usually been relegated to the oft-neglected dimension of neuroticism. It is thus possible to take account of the fact that, in addition to being differentiated in terms of extraversion, hysterics and dysthymics are, unlike "normals", also extreme on neuroticism, yet are characterized clinically by different levels of manifest anxiety.

It is not surprising, perhaps, that these differences between normals and neurotics, as a whole, and between the two neurotic syndromes, are not without influence on tests of the sort used to investigate the excitation–inhibition hypothesis. The difficulties encountered previously lay mainly in conceiving a theoretical model whereby, although extraversion and neuroticism were statistically orthogonal dimensions, the total behaviour they described could be visualized within the common framework of the

excitation–inhibition hypothesis. Consideration of recent work in neurophysiology helped in this respect, by elucidating some of the possible physiological correlates of excitation–inhibition, and pointing to a common link with the concepts of current arousal theory.

In developing our arguments here we have used the term "dysthymia–hysteria" in a relatively restricted sense, taking the anxiety state and the conversion hysteric as cases typically falling at each end of that continuum. It is true, of course, that Eysenck (1960a) with the support of Hildebrand's results (1958), has subsumed other diagnostic categories under the general heading of dysthymia and hysteria. Obsessionals and neurotic depressions are considered to fall at the dysthymic end, while psychopathy is placed with hysteria. Storms (1958) has criticized this analysis, particularly with regard to the combining of hysteric and psychopathic groups, while Slater (1960) has recently attempted a synthesis of the findings of Storms and Hildebrand. It would be irrelevant to our main point here to consider the validity or otherwise of these factor analytical studies of dysthymia–hysteria. Suffice it to say that there is sufficient experimental support for Eysenck's general classification of neuroses, as, for example, in the sedation threshold results of Shagass (Shagass and Jones, 1958), whose various diagnostic groups fell very much in the order predicted by Eysenck from his excitation–inhibition hypothesis. On the other hand, no one working in this field could deny the need to refine the rather broad category of dysthymia–hysteria by investigating neurotic groups other than those used here. It would indeed be remarkable if no differences were found between such groups. In this paper, however, we must content ourselves with a few preliminary, and sometimes disjointed, thoughts on this problem, most of which it would be difficult at present to incorporate directly within the theoretical framework we have developed.

In discussing psychiatric abnormality, we have assumed that all neurotics, whether dysthymic or hysteric, are characterized by extreme autonomic reactivity. They are visualized as forming the upper end of a normal distribution, the other extreme of which would contain an equally small number of individuals who were extremely *unresponsive*. The normal population would tend to fall, therefore, midway between these extremes, showing not low but moderate reactivity. It is possible that the psychopath falls in this extremely unresponsive group, a physiological status which would be as "abnormal" as extreme autonomic reactivity.* This, together with the high level of cortical inhibition with which he has been characterized, would, on two accounts, put the psychopath in the most unfavourable position with regard to ease of conditioning. On the other hand, that psychopaths are a heterogeneous group in this respect has been indicated by Tong (1959), who found some psychopaths to be overreactive.

With regard to hysteria, we have tended to concentrate here on the possible mechanisms underlying the more classical hysterical conversion symptom. There are, however, other forms of neurotic reaction which are commonly classified under the general heading of hysteria, e.g. the histrionic behaviour

* Essentially the same point has recently been made by Walton (1960).

of the immature hysterical personality. While at present it is difficult to evaluate at a neuropsychological level, it is worth noting the analysis of emotion in terms of "anger in" and "anger out" (Funkenstein *et al.*, 1954), and the evidence associating anxiety with adrenaline, and anger with noradrenaline secretion (Gellhorn, 1957; Cohen and Silverman, 1959; Ax, 1953). With regard to the latter point, it is interesting, therefore, to note: (a) the similarity between the "acting out" type of behaviour found in the hysterical personality (and possibly some psychopaths), the temper tantrums of the infant, and the uncoordinated rage response of the neonate, in whom noradrenaline level is found to be high (West *et al.*, 1951); and (b) the finding of Weil-Malherbe *(op. cit.)* that plasma noradrenaline level was highest in his combined hysteric–psychopath group.

While considering Weil-Malherbe's results, it is also interesting to note that adrenaline levels were highest in depression, while obsessionals had only moderate levels of both adrenaline and noradrenaline. With regard to the latter finding, it is perhaps not without significance that obsessionals are also found to have only moderate sedation thresholds, both in Shagass's work (Shagass and Jones, 1958) and in the case of the few we ourselves have been able to test. If we were to attempt an evaluation of these various findings, it would be in the following tentative way.

Neurotic reactions may be thought of as attempts to deal with stress situations which give rise to anxiety, presumably occurring in association with an increase in adrenaline level. The different neurotic syndromes reflect, therefore, the various ways of dealing with these situations. We have already analysed in some detail how the conversion hysteric may deal with stress. Paradoxically, the obsessional may be thought of as having some similarity with the conversion hysteric, in the sense that his compulsive symptoms convert or remove his need to feel anxious. The two differ, significantly, in so far as the conversion hysteric makes use of a massive inhibitory effect, while the compulsive acts of the obsessional are the result of overlearned habits. In the case of hysterical personalities, conversion of anxiety is achieved by the acting out of emotional arousal,* thus possibly accounting for the predominance of noradrenaline in these patients. By contrast, minimal attempts at dealing with anxiety are made by the anxiety state, in whom emotional arousal persists at a high level, accompanied by somatic symptoms. If we were to guess at the position in this scheme of neurotic depression, it would be that this condition represents the extreme (high) end of the continuum of emotional arousal. The inability to deal effectively with increased anxiety and the consequent inactivity of the depressed patient may, therefore, be analogous to the effects illustrated in U-shaped relationships between performance and drive, i.e. a reduction in response once arousal is taken beyond an optimum point.

Much of what has been said in the previous paragraphs must remain pure speculation until further work is undertaken. Nevertheless, one conclusion seems inescapable. That is that although we, in common with other

* Note in this respect the possible relevance of Freeman's "discharge control" factor (1948).

workers in this field, have felt the need to emphasize the role played by autonomic factors in neurosis, this should not blind us to the probability of individual variations at other levels of nervous function, as suggested in the cortical inhibition hypothesis of Eysenck. Indeed, the study of various neurotic syndromes, at present subsumed under the heading of dysthymia-hysteria, may throw further light on those nervous processes underlying different attempts to deal with basically similar changes in autonomic arousal level.

In conclusion, perhaps we should apologize to those readers who feel that this paper has been used as a vehicle, rather for developing personality theory, than for elucidating some of the more general principles of psychopharmacology. That we should have felt it necessary to make such an apology, itself underlines the close link between these two areas of behaviour. Indeed, it is surprising that this link, so long informally recognized by psychologists, physiologists and clinicians, should only in recent years have been subjected to careful experimental study.

Chapter 6

A NEW METHOD FOR THE DETERMINATION OF INDIVIDUAL DIFFERENCES IN SUSCEPTIBILITY TO A DEPRESSANT DRUG

EMER RODNIGHT and R. N. GOOCH*

A resurgence of interest in the relationship between susceptibility to depressant drugs and individual personality differences has occurred during the past seven years. Whilst the Shagass "sedation threshold" method provided a promising tool for the investigation of this relationship (Shagass, 1954) Eysenck's stimulating analysis of differential drug susceptibility in terms of differences in central inhibition levels offered a broad theoretical framework for further experimental study of the problem (Eysenck, 1955; 1957a).

The progress that has been made since 1954 has been extensively reviewed elsewhere (Claridge and Herrington. Chapter 5): on the positive side, confirmation has been obtained for the general proposition that degree of susceptibility to a depressant drug is significantly related to differences in extraversion, although the relationship now appears to be more complex than had at first been thought (Shagass and Kerenyi, 1958; Claridge and Herrington, 1960); equally important, though on the negative side, has been the discovery of serious practical difficulties in the application of the original "sedation threshold" method, resulting in its failure to meet an acceptable criterion of reliability (Thorpe and Barker, 1957; Ackner and Pampiglione, 1958).

Whilst Shagass himself expressed confidence in the determination of sedation thresholds by electroencephalographic measures, he nevertheless had some doubt about the practical applicability in clinical diagnosis of his somewhat elaborate technique and has therefore more recently introduced a "sleep threshold" method which utilizes a purely behavioural criterion of susceptibility to sedative drugs (Shagass, 1959). Claridge and Herrington (1960) similarly argue that for research purposes also, the replacement of the rather unwieldy EEG technique by some simpler behavioural method would be practically desirable, provided that a reliable and valid behavioural criterion could be devised. They adopted a method for assessing sedation thresholds in terms of the effect of Sodium Amytal on a simple oral arithmetic task. Subjects received a continuous intravenous infusion of Sodium Amytal at the rate of 0·1 G/min. At the same time they were asked to respond to a tape-recorded random sequence of single digits by doubling

* The authors gratefully acknowledge the financial support of the U.S. National Institutes of Health, in carrying out this work and a Fellowship of the Mental Health Research Fund to one of us (R.N.G.).

each digit as it occurred, and calling out the answer in the 2 sec period before the next digit was presented. Responses to this task were divided into successive blocks of five, the sedation threshold being defined as the point midway between the last two blocks with less than 50 per cent error and the first two blocks in which errors exceeded 50 per cent. The amount of drug administered at this point was determined, and expressed in terms of mg/kg body weight. Using this technique, the authors report highly significant discrimination between groups of neurotic patients diagnosed as dysthymics and a normal control group, and also between dysthymics and neurotic patients diagnosed as hysterics. The technique is less successful in discriminating between normals and hysterics although the latter group have the lower mean sedation threshold, as expected.

Methodological Considerations

Commenting on the negative results in the Ackner and Pampiglione study (where the Shagass sedation threshold test failed to discriminate between neurotic groups differentiated in terms of anxiety ratings), Claridge and Herrington justifiably suggest that their failure may have been due to "the uncertain nature of their sedation threshold measure (rather) than to a real difference between these (neurotic) groups." *(op. cit.*, p. 1569). One of the main aims of this investigation (Ackner and Pampiglione, 1958) was, in fact, to determine the reliability of the criterion measure and, although they also report dissatisfaction with the anxiety rating method used in classifying their neurotic groups (cf. Shagass and Naiman, 1956), their main conclusion is that the reliability of the sedation threshold measure used (as assessed by extent of agreement between independent judges of the test records) is unacceptably low.

The utilization of a standardized behavioural test to determine sedation thresholds (Claridge and Herrington, 1960) makes it possible to reduce the unreliability of the scores substantially, by eliminating the possibility of inter-judge disagreements on the scoring of test records. Other sources of unreliability may of course remain, and although some such sources may fairly easily be discovered and brought under control, it seems certain that others will remain undetected and uncontrolled for some time to come.

Closely related, though not identical with the problem of reliability, is that of the degree of precision of the sedation threshold determination. The level of accuracy required will vary, of course, in accordance with the purposes for which the determinations are made, but for clinical diagnostic purposes and predictive experimental studies alike, the required degree of precision may be very high. It is perhaps significant that the need for careful scrutiny of this aspect of the score is particularly clearly demonstrated in the work of Claridge and Herrington *(op. cit.)* where the problem of score unreliability has been so successfully tackled.

Their Fig. 1 (reproduced in Chapter 5.) demonstrates that the hysteric subject reached his sedation threshold point approximately 4 min after the beginning of the test, and the normal subject 1 min later. The difference between these two subjects in amount of Sodium Amytal administered would therefore be 100 mg. Since the criterion used in assessing

the individual's sedation threshold involves a minimum period of 40 sec, during which 66·6 mg of drug is administered, the question arises whether the particular drug and technique of drug administration employed may not produce a behavioural deterioration which is too precipitate to be charted sufficiently precisely by the behavioural criterion chosen to measure it.

The problem here, in its most general form, is that of deciding which of a number of alternative behavioural measures will prove to be the most capable of mirroring the physiological deterioration function which is to be assessed. In designing studies of this nature, which involve the determination of differential susceptibility to a drug in terms of the amount of drug required to produce a behaviourally defined level of deterioration, two of the crucial considerations appear to be: (a) the degree and precision with which effective drug levels can be maintained and varied over time; and (b) the potential "information capacity," per unit of time, of alternative behavioural measures. Drugs that differ in their speed of action will almost certainly demand the application of different criteria of susceptibility; Shagass, for example, assesses differential susceptibility to the fast-acting Pentothal and the slower-acting Sodium Amytal by applying quite different criteria (Shagass, 1954; Shagass, Muller and Acosta, 1959). It may be that at our present stage of knowledge and skill, the least time-consuming techniques are also least useful for research purposes, because the small space of time available suggests the need for a highly sensitive and discriminating criterion, but at the same time allows only a relatively crude measure to be applied. Decisions concerning the usefulness of alternative drugs and alternative behavioural tests for the measurement of differential susceptibility to sedation must still be largely arbitrary, or guided by the results of trial and error. An analysis of Claridge and Herrington's results *(op. cit.)* was instrumental in suggesting the alternative techniques adopted by the present authors in the investigation to be reported here.

One further aspect of the problem of test reliability remains to be considered; this is the question of whether, or under what circumstances, it is justifiable to regard the comparison of test and retest results as a valid indication of the reliability of sedation threshold determinations. Test-retest data are scarce in the literature. Shagass, however, whose sedation threshold technique was earlier reported to yield highly stable results on retest (Shagass, Mihalik and Jones, 1957), has more recently reported some interesting changes between test and retest sedation thresholds (Shagass, 1958), and sleep thresholds (Shagass, Muller and Acosta, 1959) in neurotic patients. He has examined these threshold differences in relation to changes in the patients' symptomatology resulting from electroconvulsive therapy, and reports correlations ranging from 0·57 to 0·90.

Interpretation of these correlations as reliability coefficients is difficult because of the complexity of the conditions under which they were obtained. Although the measure of agreement is encouragingly high, in view of the less than optimal conditions prevailing throughout this series of investigations, it must nevertheless be concluded that neurotic patients undergoing therapy are not the most suitable subjects for the establishment of test-retest reliabilities as such.

Since there is no necessary incompatibility between high reliability in a measuring instrument and large discrepancies between the values obtained on successive applications of the instrument, it would seem premature to conclude that the Shagass test-retest studies constitute evidence for the unreliability of his techniques (as some critics have done). In the absence of any certain independent evidence that the sedation threshold measure is in fact reliable (Shagass reports the results of inter-judge reliability checks in some of his studies), test-retest discrepancies may of course, equally well be due to lawful changes in subjects' susceptibility to the drug administered, to unreliability of measurement, or to both or neither of these alternatives. The dilemma, it seems, can best be solved by separate but parallel investigations rather than by piecemeal attack on several aspects of the problem simultaneously.

Among the more obvious factual problems which must be taken into account in any investigation of this kind, utilizing behavioural tests, are the following: (a) The range and direction of test-retest differences in the performance measure under normal conditions of test administration not involving the use of sedative drugs, and the influence which varying the time interval between tests may have on the magnitude of any learning or practice effect that may be present; (b) The extent to which subjects may be able to learn compensatory techniques for the control or concealment of behavioural deterioration induced by the sedative drug, in the test situation; (c) The extent to which inter-drug generalization of the type of learning described in (b) may occur. Is it possible, for instance, that a change in the frequency of habitual consumption of alcohol occurring in a normal subject between test and retest, may result in a general loss or gain in skill at controlling the behavioural effects of depressant drugs, which may be reflected in test performances; and (d) Changes in the state of general health, in diet etc. are less likely to produce a significant shift in the sedation threshold (except where these changes are extreme) than a change involving the introduction or withdrawal of any drug known to affect the central or autonomic nervous system directly. Ideally, since so little is known with certainty about the physiological effects of different combinations of drugs, the possibility of any complication of this kind should be competely eliminated, or the factor experimentally controlled. In practice this ideal can never fully be attained. It seems worthy of mention nevertheless, because the literature indicates that some investigators may have been too little concerned with the possible effects on their test results of interactions between the test drug and previous medication, and have consequently failed to make use of whatever relevant information was available.

The preceding discussion suggests the general conclusion that, as we are still far from completing the task of sorting out the fortuitous and transient from the lawful and relatively stable influences which cause changes in susceptibility to depressant drugs between test and retest, some alternative method of establishing measurement reliability as such is to be preferred.

At the present stage of development of research in this field, it would be a mistake to pursue the goal of high statistical reliability too assiduously

or too exclusively. Although it is generally accepted that the practical validity of a test is directly proportional to its reliability, these two goals may sometimes be incompatible (cf. Guilford, 1956, Ch. 18). This is expecially likely to be true where the function to be measured is heterogeneous and involves a variety of interacting or co-acting components. Whether one examines drug susceptibility from the point of view of the neurophysiological and biochemical mechanisms involved or from the point of view of its behavioural manifestations, the indications are equally strong that the most valid and sensitive differential tests will prove to be multidimensional tests with relatively low intercorrelations between test components. The individual is known to be richly endowed with equipotential compensatory mechanisms for dealing with any influence which threatens to upset the balance of the "*milieu interieur*," and in Chapter 5 of this book, Claridge and Herrington have outlined the variety of alternative central and autonomic nervous system processes that are potentially capable of maintaining or re-instating any given state of nervous equilibrium. In view of the evidence for an equally wide range of inter-individual variation in the manifestation of functionally equivalent behavioural epiphenomena (cf. Steinberg, 1956), it is not so surprising that such simple indices of susceptibility to depressant drugs as the slurring of speech or the onset of lateral gaze nystagmus have proved unsatisfactory (Fink, 1958; Shagass and Naiman, 1956; Thorpe and Barker, 1957).

Aims of the Present Study

This investigation was instigated in the belief that previous work concerning the applicability of sedative tolerance tests in psychiatric diagnosis and the relationship between susceptibility and personality structure, had produced sufficiently strong evidence in support of the theories of Eysenck and Shagass to suggest that the conflicting results reported by other investigators might be resolved, if an improved technique for assessing susceptibility could be developed. We aimed, therefore, to devise an assessment technique which would utilize unambiguous objective criteria with reasonable face validity, and then to test the replicability of the measures obtained and the relationship between these measures of drug susceptibility and the personality variables of extraversion and neuroticism.

Description of Subjects

Thirty-two normal volunteer subjects participated in the experiment of whom 4 were research workers in the Department of Psychology. The remaining 28 subjects had occupations unconnected with psychological or medical research and were paid for their services. All subjects, with one exception, were male. Ages ranged from 17 to 47 years, the mean age being 26·0 years $\pm$6·5. Standard I.Q.s were not obtained for all subjects, but the average I.Q. estimated to be about 120, or higher than one standard deviation above the population mean.

The records of 7 subjects are not included in the results to be reported; 4 (including 2 of the unpaid volunteers) performed a variety of behavioural

tests which were tried out in the initial stages of the investigation but later discarded as unsatisfactory. The records of 2 other subjects were rejected because they altered the position of the breathing mask during the experimental session, causing an unknown quantity of gas to escape; one subject complained of nausea soon after the administration of nitrous oxide was begun and the test was therefore discontinued.

Every subject was asked to complete the Maudsley Personality Inventory (Eysenck, 1959), usually on the first day of testing and before any other tests were introduced. If subjects returned for retesting more than 2 months after their first appointment, they were asked to complete the Inventory a second time; otherwise only one set of personality scores was obtained. The Personality Inventory data in Table 1 do not include any retest scores.

TABLE 1

Distribution of extraversion and neuroticism scores (M.P.I.) in the experimental sample

n = 25	*Extraversion*	*Neuroticism*
Range	6–41	1–43
Mean	26·21	26·38
Standard deviation	8·05	12·11

Selection of a Depressant Drug

A nitrous oxide in oxygen mixture was chosen as a suitable general central nervous system depressant for several reasons. The similarity of the modes of action of different general depressants (discussed in Chapter 15 of this book) is such that it was thought justified to use nitrous oxide rather than Pentothal or Sodium Amytal as in previous work on the Sedation Threshold. After reviewing the results obtained in a number of studies where one or other of these two drugs was administered, Shagass concludes that a further extension of the range of drugs and of tests would probably add much to our present knowledge about the relationship between drug susceptibility and personality (Shagass, Muller and Acosta, 1959). It was felt that with this depressant a more accurate control over the cerebral content of the drug could be maintained than with the use of intravenous barbiturates, and moreover, since the necessity for venipuncture would be eliminated and the recovery time be shorter, the procedure would be more acceptable to subjects.

Nitrous oxide is an inorganic gas with a slightly sweetish odour. It does not enter into chemical combination within the body and is absorbed and eliminated through the lungs (Lee, 1959). The relationship between the percentage of nitrous oxide in the inspired gas mixture and the percentage present in the alveolar air of the lungs depends on the mechanics of respi-

ration and the physical laws governing mixtures of gases and vapours; similarly the relationship between the drug concentrations in the alveolar air and in the blood of the pulmonary capillaries is determined by the laws governing diffusion and solution of gases in liquids (Lee, 1959; Wilson and Schild, 1959).

The rate of carriage of nitrous oxide to the brain depends on circulation time, and the rate at which the gas accumulates in the cerebral tissues depends mainly upon the vascularity of the organ and the rate of blood flow through it. These again follow the physical laws concerned with the solution and diffusion of gases in liquids. Whilst for any particular level of gas it may take several hours for the bodily tissues to become saturated and reach a stable equilibrium with the inspired gases, we were only concerned with building up a partial equilibrium between the concentration of gas in the arterial blood and the cerebral tissues. This can be attained in a relatively short space of time owing to the extreme vascularity of the cerebral tissues (Wilson and Schild, 1959).

Sources of individual variation between a given concentration of inspired gas and cerebral content will therefore depend mainly on differences in pulmonary efficiency and rate of carriage in the blood. The longer the period of time, however, over which a particular level of gas is inhaled, the more nearly an absolute equilibrium is approached and the less the

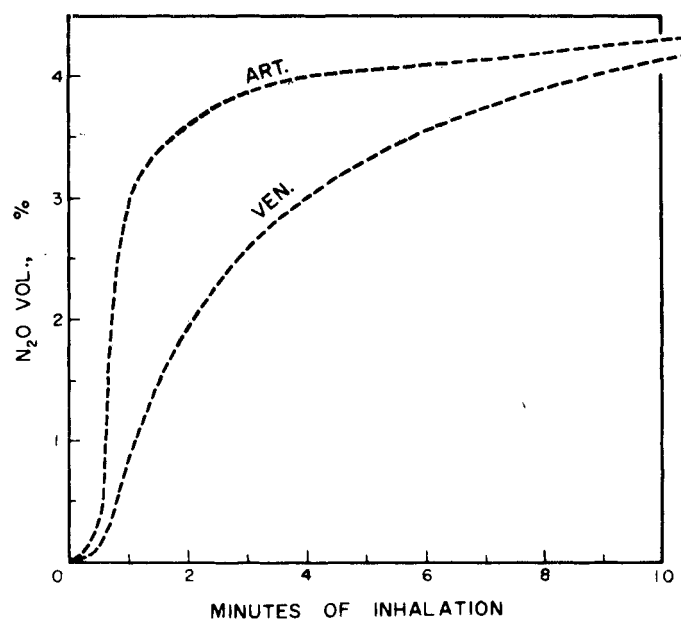


FIG. 1. Typical arterial (Art.) and internal jugular (Ven.) curves of N_2O concentration during a 10 min period of inhalation of 15% N_2O. (Reproduced with permission from Kety and Schmidt, 1948a).

inter-individual variation in level of cerebral nitrous oxide. Despite the fairly rapid rise in the cerebral concentration during the first minute after nitrous oxide is introduced, there is apparently little concomitant behavioural effect, as has been demonstrated, for example, by Holland (Chapter 3) with respect to the effect of this drug on a visual phenomenon. (There was little drug effect during the first minute, a build-up to a maximum effect during 5–6 min after the introduction of the gas, and a decline in apparent drug effect thereafter which was possibly due to adaptive mechanisms.) To have aimed at the achievement of complete equilibrium between the concentration of nitrous oxide in brain tissues and in the inspired gas mixture would have been impractical within the framework of the experimental design. As there were medical objections to the exposure of subjects to moderately high concentrations of nitrous oxide for a long period of time, a choice had to be made between the investigation of either a small number of dosage levels with a 10 min inhalation period at each level, or a larger range of dosages, each for a shorter period. From the point of view of attempting to define the deterioration function as such, the latter alternative seemed preferable. Five minute periods of exposure to various gas concentrations were, therefore, adopted, since it has been shown that at the end of 5 mm, equilibrium between the drug concentrations in the blood stream and the brain is already more than three-quarters established (Kety and Schmidt, 1948a).

Selection and Description of Behavioural Tests

After initial decisions had been made to limit the total time of inhalation of dilute nitrous oxide to a maximum of 50 min, and within that time to increase the concentration of the gas in small steps at 5 min intervals, various behavioural tests were selected that could be administered within the time limits thus imposed. The next major consideration was that the test or tests should be sufficiently complex to yield a fairly large, and finely graded range of scores. With respect to level of difficulty, the problem was to reach a satisfactory compromise between a too difficult task (where a maximal failure score might be reached too soon, and where performance might correlate highly with intellectual ability) and a too easy task in which significant deterioration in performance would occur only at high levels of drug dosage. The final selection of tasks was based on Steinberg's ranking of cognitive tests in terms of differential impairment of performance under the influence of a 30 per cent concentration of nitrous oxide in oxygen (Steinberg, 1954).

(a) *The motor task* — Similar to the test most readily affected by nitrous oxide in Steinberg's battery, the motor task employed in the present experiment is a modified version of the Pegboard Finger Dexterity test in the General Aptitude Test Battery (U.S.E.S., 1947). The subject was required to move small, round-headed metal pegs from retaining slots in the lower half of a rectangular wooden board to corresponding slots in the upper half of the board. The pegs (of which there are 50) were moved one at a time in a set order. Subjects were instructed to use the preferred

hand only, right-handed subjects starting from the right-hand side of the pegboard and working to the left, and vice versa for left-handed testees. In the preliminary instructions, subjects were also told how to proceed with the test if they succeeded in transferring all 50 pegs to their new positions within the 1 min time limit; the pegs would then be moved back to their original positions, one at a time. Uncontrolled variations in score resulting from time lost in trying to retrieve pegs dropped in transit were minimized, by forbidding the replacement of these pegs during the test. The score was the number of pegs correctly moved in the course of a minute.

(b) *The arithmetical task* — Two associative tests involving response to numbers were ranked midway between the most and the least impaired by nitrous oxide, in Steinberg's battery of cognitive tests (Steinberg, 1954). These were: (a) "Arithmetic," the addition of columns of 2-digit numbers, and (b) a standard immediate memory test, "Digit Span, Backward." It was thought that a test comparable with either of these might possibly provide a more discriminating measure of drug susceptibility at high dosage levels than would the motor task. The test initially adopted was the simple multiplication task previously used by Claridge and Herrington (1960) in the determination of Sodium Amytal sedation thresholds; although requiring less "mental effort" than the Steinberg addition test, it involved the exercise of a very similar skill. An additional recommendation for its use was the opportunity thus provided for comparing the effectiveness of the test scores as indicators of drug susceptibility when applied in quite a different experimental situation from that in which they were first found useful.

A small number of trial runs indicated that the multiplication test in its original form was not well suited to our requirements, because it yielded almost no measurable error variance until the subjects' intake of gas was approaching the upper limit of our drug dosage range. Several variations of the test were then tried out before the final adoption of the following modified version: Subjects were asked to multiply single-digit numbers by three and to call out the answer in the interval before the next test number was presented. The test material and preliminary instructions were recorded on magnetic tape and relayed to subjects through a tape-recorder amplifier. The sound volume was varied from subject to subject in accordance with individual requirements, in order to eliminate error variance attributable to differences in auditory acuity between subjects.* Test numbers were presented in 2 min blocks of 60, a new number being presented every 2 sec irrespective of the subjects' success or failure in responding to the preceding digit. Each block of 60 numbers was immediately preceded by a "Get Ready" signal and was separated from the succeeding block by a 3 min interval. The score was the number of errors in each 2 min test interval.

* Burns, Robson and Welt (1960) have reported significant increases in auditory thresholds under the influence of 25 percent nitrous oxide in oxygen.

EXPERIMENTAL PROCEDURE

After a preliminary medical screening, each subject was taken to the testing room where the general plan of the experimental session was outlined for him. He was hown the various pieces of test equipment and was encouraged to ask questions about his part in the experiment. Not unexpectedly, the most frequent queries were those concerning the kinds of subjective symptoms likely to be experienced under the influence of the drug. Nitrous oxide was already familiar to some subjects as a dental anaesthetic, and particular care was taken to assure these and other members of the experimental group that the concentrations to be used fell far short of an anaesthetic dose. To forestall the possible occurrence of panic reactions during the testing session and to help reduce apprehension in general, subjects were forewarned about the usual subjective effects of the gas. It was pointed out to the subject that he could communicate with both the experimenters throughout the testing session and that the test could be discontinued immediately, at any stage, should it become unpleasant for him to proceed.

A period of preliminary practice always preceded the main test session. An average of two 2 min trials was found sufficient to familiarize subjects with the multiplication test and to obtain one error-free trial. Practice on the pegboard test was continued in 1 min trials, separated by 4 min rest periods, until performance showed no further improvement. Successive trial scores followed a pattern typical of the learning of a simple motor skill, with a rapid rate of improvement in the early trials followed by a decelerated ascent to a plateau of performance. The average number of practice trials was 7, the range for the group being from 4 to 12 trials. A comparison between the first- and second-occasion final trial scores of subjects who had been re-tested after varying periods of time tended to confirm that performance on the Pegboard task had reached asymptote in the first pre-rest practice session; there was no significant difference between the two sets of final pre-test practice scores.

Throughout the initial practice period as well as during the experimental session proper, the subject was seated in a comfortable low chair; the apparatus for administering the gas mixtures was placed behind him so that he would be unable to observe the manipulation of the controls or identify the gases being used at any particular time. (Although ethical considerations required that subjects should know that nitrous oxide would be used sometimes during the experiments, they were not told exactly when it was to be introduced nor given any information about placebo runs.) The main experimental session was usually begun a few minutes after the completion of the practice period; the breathing mask was adjusted to fit the subject's face as closely and comfortably as possible; the Pegboard test was then placed in front of him on a tray and he was given a final reminder about the sequence of tests and rest periods.

The gas mixtures were administered with a Boyle Anaesthetic Apparatus as supplied by the British Oxygen Company. The measurement of the proportions of gases in the mixture was done by means of the standard

Rotameter flowmeters supplied with the machine. The gas mixture was delivered to the subject by an "open circuit" technique (the expired air being discharged via the expiratory valve of the mask into open atmosphere) and a mask having as little as possible "dead space" below it was chosen in order to minimize any possible reinspiration of the carbon dioxide in the exhaled air. The mask was fitted with a microphone through which the subject could maintain verbal communication with the experimenters.

Subjects were given a preliminary period of 10 min breathing oxygen alone after which nitrous oxide was introduced.* (although a 10 min exposure to an atmosphere of pure oxygen would produce a significant decrease of about 13 per cent in mean cerebral blood flow, there would be no change in cerebral oxygen consumption (Kety and Schmidt 1948 b). The percentage of nitrous oxide in the nitrous oxide–oxygen mixture was increased in stepwise fashion every 5 min until the desired range of percentage mixtures was covered. The method used for placebo test sessions was exactly the same, except that compressed air of medical quality was used in place of nitrous oxide. No attempt was made to disguise the smell of the gases because the addition of a volatile aromatic substance, perhaps having a biological action of its own, was not considered justified when the subjective symptoms experienced by subjects inhaling nitrous oxide could not themselves be disguised.

The testing was carried out so that the arithmetic task was given during the last 2 min of each 5 min "step" of gas level and the motor task during the first minute of the succeeding "step" (during which it was considered that the operative level of nitrous oxide would not change substantially from that attained during the preceding minute). Thus there were 3 min of testing and 2 min of rest during each 5 min interval.

RESULTS

Finger Dexterity Test

The effect of administering increasing concentrations of nitrous oxide in oxygen on the performance of individuals in this test was reflected generally in a small early drop in the speed of performance, followed by a gradual proliferation of other behavioural signs of impairment including fumbling, dropping pegs and under- or overshooting the target slots.

The kind of graph which resulted from plotting the raw scores for each subject against the appropriate gas concentrations at each stage of testing was usually a curvilinear one. When the values of the percentage concentrations of nitrous oxide were transposed into units based on aninverse logscale (as shown in Table 2) and the raw scores then replotted, the relationship between performance and drug dosage became apparently rectilinear. The results for 3 individuals with contrasting performance trends are illustrated in Fig. 2.

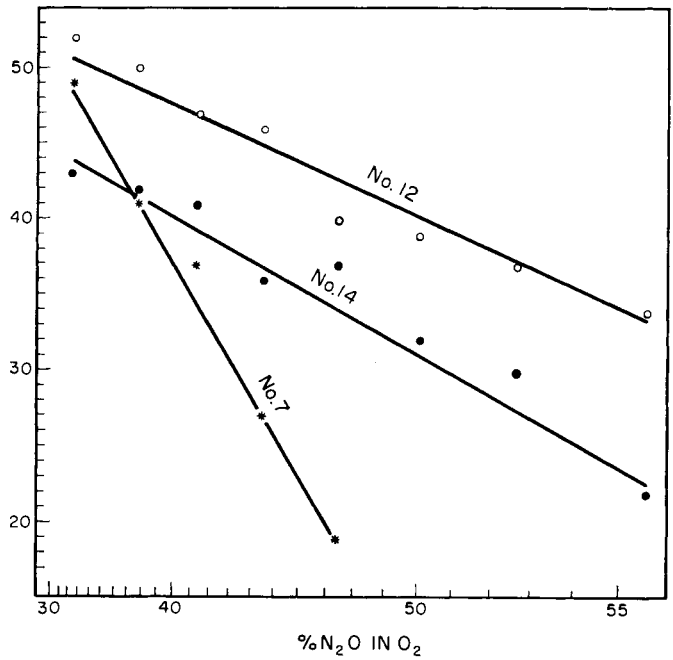


FIG. 2. Performance scores obtained by 3 subjects on a finger dexterity test during inhalation of various concentrations of N_2O in O_2. Performance levels are indicated on the ordinate and transformed* drug dosages on the abscissa. (*For details see text.) Lines of best fit are drawn through each set of scores.

TABLE 2

Transformation of N_2O Concentration Values

Percentage of Nitrous Oxide in Nitrous Oxide–Oxygen mixture	*Units used on axis (cf. Fig. 3)*
0	0
33·3%	45·4
37·5%	62·26
41·48%	81·76
44·4%	103·8
47·37%	128·2
50%	155
52·63%	187

It was decided to test the rectilinearity of this relationship, using the performance scores of the first 15 subjects over the dosage between 33 per cent and 47·27 per cent nitrous oxide.

The rectilinearity of the relationship between performance on the motor test and appropriate gas levels was tested by deriving the statistic ϵ^2 from the correlation ratio ϵ derived in turn from a simple two-way analysis of variance. (The method is described fully in Edwards (1946) (p. 237 foll.). This statistic ϵ^2 was found to have a value of 0·008 which is well below the 5% level of significance and therefore indicates that the departures from rectilinearity in the relationship are insignificant. The rectilinearity of the relationships between performance scores and drug dosage levels for each individual is indicated by the correlation coefficients derived from these measures for each subject. This statistic (r) for each of 11 of the 15 subjects lay between the values of −0·99 and −0·91 ($p < 0·05$), whilst for the remaining 4 subjects (r) had values of −0·804, −0·582, −0·405 and −0·372. Thus for the majority of individuals in the group, there was no significant deviation from the rectilinear function[1].

The next step in the consideration of the Pegboard test was to derive a score for each of the subjects who performed it (23 in all) which would serve as a reliable index of the individual's susceptibility to nitrous oxide. This was done by taking the raw scores for each individual, fitting a line of best fit to each set of data and thereby deriving the expected decrement for each individual at the 50 per cent gas level in terms of a percentage of his theoretical starting level. As these percentages were distributed in a skewed fashion, a logarithmic transformation was performed to give a reasonably normal distribution. The same process was repeated on the data for 9 of the subjects who had a retesting at a later date. (The interval between test and retest varied from a few days to approximately a year.) The scores thus derived for these 9 subjects show a correlation of 0·911 between test and retest, which is significant beyond the 0·001 level of confidence. A correlation of this magnitude seems to indicate that the function being tested is a relatively stable one.

The type of decrement function involved — It has already been noted above that if the drug effect (decrement in performance in terms of raw scores) is plotted against dosage (concentration of nitrous oxide), the result appears as a curvilinear function which is positively accelerated, as in Fig. 7 (Chapter 14). This differs markedly from the usual kind of drug effect–dosage curve familiar to the pharmacologist (cf. Fig. 8, Chapter 14) which is negatively accelerated and resembles a cumulative frequency distribution curve. The trend of our results would thus suggest that the effect with which we are dealing represents a progressive interference with the functioning of a system in which there is a high order of interaction between the units of which it is composed, rather than the cumulative effect of the drug on a population of independent cells with various degrees of susceptibility. This result is consistent with the physiological theories

[1] An alternative method for testing the rectilinearity of such functions is described by Gooch and Slater (In Press)

concerning the action of general central nervous system depressants on the reticular formation of the brain.

Relationship between gas susceptibility and personality measures — Extraversion (E) and neuroticism (N) scores for all subjects, obtained from their responses on the Maudsley Personality Inventory, were used to examine the relationship between these two personality variables and susceptibility to the depressant drug. When it was found that the overall correlations between gas susceptibility and E and N respectively, were insignificant, the sample was then divided into four separate personality groupings as follows (1) group I comprised those subjects whose questionnaire scores placed them above the means for the whole sample in both extraversion and neuroticism; (2) group II consisted of those who were above the mean for extraversion but below the mean for neuroticism, (3) in group III the reverse was true; and (4) individuals in group IV were below the means for both extraversion and neuroticism.

It was found that among subjects with low neuroticism scores, the correlation between extraversion and susceptibility to gas was negative and large ($r = -0{\cdot}72$ $p < 0{\cdot}05$), whilst the extraverted section of the total sample, on the other hand, exhibited a highly significant positive relationship between measures of neuroticism and susceptibility to gas. As there thus appeared to be a complex interaction between these three variables, a diagrammatic analysis of the differential incidence of susceptible, moderately susceptible and resistant subjects within the various

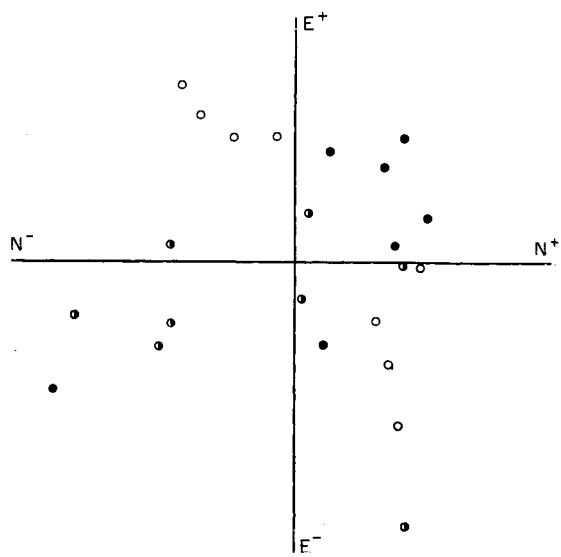


FIG. 3. A diagrammatic representation of the distribution of the total sample with respect to the personality variables of extraversion (E) and neuroticism (N), showing the interrelationship between these and degree of susceptibility to N_2O. Black circles indicate high susceptibility, half-shaded circles moderate susceptibility and white circles insusceptibility.

areas of the two-factor space defined by E and N was examined, for confirmation of interactions between the variables. Fig. 3 is a reproduction of this analysis.

Further confirmation for the tendency for susceptibility to the drug to depend on both the degree of extraversion and of neuroticism exhibited by the individual, was obtained by computing the correlation between the susceptibility index and the product of the E and N scores for the entire sample. This yielded a coefficient of 0·59 which is statistically significant ($p < 0{\cdot}01$). In Table 3, the full set of correlation coefficients is reported (including two where the absolute differences between standard scores of susceptibility and one of the personality variables, have been correlated with corresponding standard scores on the second personality dimension — an alternative way of demonstrating interaction between the variables). Unfortunately the size of the group is not sufficient for a more complex statistical analysis that might elucidate further the nature of the relationship.

TABLE 3

Relationship between the Index of Susceptibility to N_2O (G) and Personality Variables (E and N)

Sample Size and Description	*Correlated Variables*	*r.*	*Significance*
Whole Group: n = 23	G×E	0·036	N.S.
Whole Group: n = 23	G×N	0·020	N.S.
Whole Group: n = 23	G×Product of E and N (expressed as z scores)	0·590	$p < 0{\cdot}01$
Whole Group: n = 23	E×Absolute difference between G and N (expressed as z scores)	−0·689	$0 < 0{\cdot}001$
Whole Group: n = 23	N×Absolute difference between G and E (expressed as z scores)	−0·244	N.S.
E+Group: n = 11	G×N	0·802	$p < 0{\cdot}01$
E−Group: n = 12	G×N	−0·421	N.S.
N×Group: n = 14	G×E	0·398	N.S.
N−Group: n = 9	G×E	−0·719	$p < 0{\cdot}05$

Nevertheless at least three conclusions can be drawn: (1) that among members of the group having a low N score, the "introverts" tend to be more susceptible to the gas than the "extraverts"; (2) among the "extraverts" of the sample, those with an N score above the mean tend to be more susceptible than others who have N scores below the sample mean, and (3) in the group as a whole, the higher the E score, the closer the relationship between gas susceptibility and neuroticism tends to be, that

is, the higher the E score, the greater the value of the N score as a predictor of susceptibility to nitrous oxide.

The interpretation of these results, until more data can be accumulated, must be somewhat speculative. If we assume, however, that resistance to nitrous oxide is a measure of the level of excitability of the central nervous system, then it must be concluded that among subjects low on neuroticism, the "extraverts" tend to have an inherently more excitable nervous system than the "introverts." In addition to this, there is also the evidence that amongst the "extraverts" of the sample, those with high N scores tend to display more susceptibility to the depressant drug than subjects with low neuroticism scores. Both findings are in keeping with much other evidence brought forward by Eysenck (1957) and others to support the idea that the neurotic extravert is characterized by a marked tendency to generate large amounts of inhibition quite rapidly. This inhibition tends to oppose excitation and thus leads to an overall reduction in behavioural excitatory phenomena.

If we consider the building up of inhibition as a protection against excessive central excitation, the general finding of the ease and rapidity with which extraverts build up inhibition, as opposed to the manner in which it accumulates in introverts, would not be in conflict with our speculation that the non-neurotic extraverts have an inherently more excitable nervous system than non-neurotic introverts. On this basis the extravert would be inherently more vulnerable to central excitation than the introvert and would therefore have a greater "need" for a rapid build-up of inhibition. The neuroticism factor has often been regarded as a drive or internal stress variable (cf. Jones, 1960); it might therefore be considered as tending to raise the basic excitation levels of both extraverts and introverts, but producing relatively more protective inhibition in extraverts for the reasons stated above. (This interpretation appears to be in keeping with certain of the findings of Russian experimentalists investigating the properties of "strong" and "weak" nervous systems. For example, Nebylitsyn, Rozhdestvenskaya and Teplov (1960) report findings indicating a higher reactivity in terms of sensitivity to peripheral stimulation in "weak" nervous systems than in the "strong" type. Their "weak" nervous system types were discriminated from the "strong" on the basis of the decrement shown in a previously well established conditioned response resulting from 10 closely repeated reinforced trials; "weak" types showed a significant decrement whilst the "strong" nervous systems did not. That is, reactive inhibition could be elicited more easily in subjects who showed a higher sensitivity to peripheral stimulation than in those who were relatively less sensitive to peripheral stimulation.) A diagrammatic representation of the suggested speculative relationship between extraversion and neuroticism is shown below (Fig. 4).

Arithmetic task – As the concentration of nitrous oxide was increased various qualitative changes in performance took place which, despite wide individual variations, seemed to follow one another in some kind of lawful sequence. The most frequent and the earliest effect of the drug was to increase reaction times to the stimulus numbers. As it was not

possible to obtain exact measures of reaction times, this change was charted in terms of the increased frequency of delayed responses and non-responses to numbers. At higher dosage levels (and mainly from the 44 per cent N_2O step onward) the general *manner* of responding changed; there was a tendency to shout rather than speak the answers and responses were frequently accompanied by a rhythmic beating of one hand on the other,

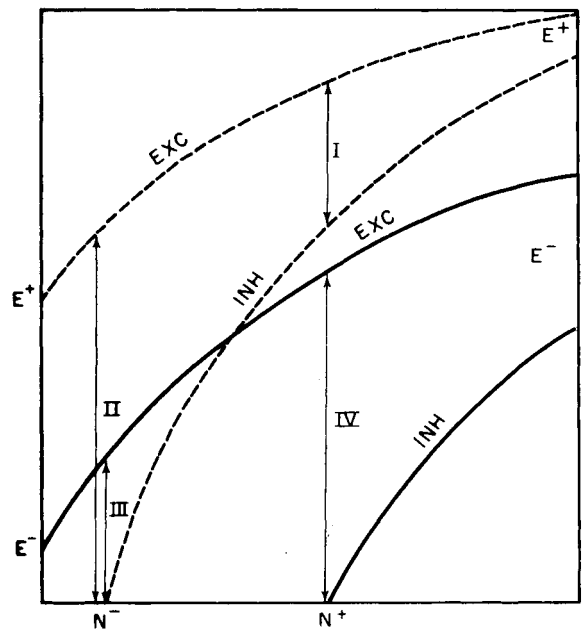


FIG. 4. Diagrammatic representation of explanation advanced in text for observed relationship between extraversion and neuroticism and an index of susceptibility to N_2O. According to this, extraverts (E+) have more excitable nervous systems than introverts (E−). Neuroticism, considered as a drive, is shown as raising excitatory potential (EXC) in E+ and E− groups; a "compensatory" inhibitory potential (INH) is thereby initiated and appears in E+ groups at lower levels of N than in E−, due to their greater need for protection against excessive excitation. N_2O susceptibility is interpreted as reflecting balance between EXC and INH. Placement of 4 personality groups in diagram (I = E+ N+, II = E+ N−, III = E− N−, IV = E− N+) is based on appropriate values of EXC−INH.

or by tapping the feet on the floor. Many subjects also reported that the maintenance of concentration on the task became impossible when they closed their eyes, looking around the room while they listened to the numbers helped them to respond more satisfactorily.

The hypothesis that hesitations in response, as well as the tendency to shout out responses in the later stages of the experimental session

might have been the results of a decrease in auditory sensitivity, was not supported by subjects' comments during and after the experiment. Although about one-third of the group reported "sounds in the ears" in the course of the testing, these were said not to interfere with hearing. From other comments about the perception of sounds, made during the experimental sessions, it appeared that the effect of the gas was not so much either to increase or decrease auditory acuity as such, but to decrease the subjects' ability of attending *selectively* to sounds (i.e. of inhibiting irrelevant auditory stimuli). If subjects' reports are reliable in indicating that their increased awareness of individual sounds inside and outside the testing room did not interfere with their ability to hear test stimuli, but actually helped them to respond by keeping drowsiness at bay, then there might be grounds for suggesting that the decline in selectivity of auditory attention is to some extent a compensatory mechanism which serves the same purpose as the increase in motor activity that accompanied verbal responses in the latter part of the test. By raising the level of stimulus input, both phenomena might serve to maintain the "tone" of the reticular activating system against the directly depressant effect of the nitrous oxide.

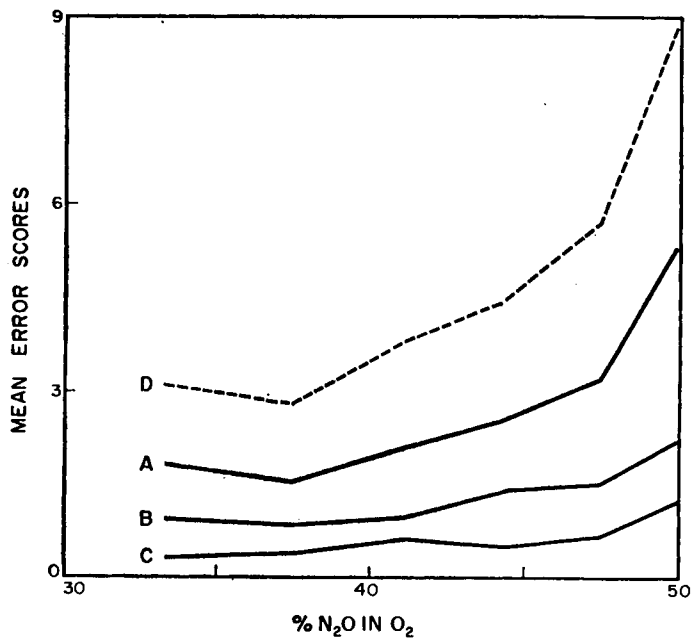


FIG. 5. Deterioration in performance of an arithmetical task under the influence of increasing concentrations of N_2O in O_2. The mean frequencies of non-responses (A), incorrect responses (B) and perseverative responses (C) for a group of 25 subjects are plotted separately. D represents the sum totals of the three sets of frequencies (mean composite errors).

As Fig. 5 shows, for the group as a whole, increases in total error within the 33–50 per cent range of N_2O concentrations tend to follow a positively accelerated growth function which is comparable with the course of deterioration of Pegboard performance. When the performance of individuals was examined, however, it was not found possible to describe the course of deterioration in terms of any common mathematical function. Under the impression that there were marked inter-individual differences in the relative frequency of occurrence of particular kinds of error, the total error score was broken down into three categories: (1) non-responses *(N-R);* (2) perseverative responses (repetitions of the stimulus number *(P-R);* and (3) incorrect responses other than perseverations *(W-R).* Whilst this analysis confirmed the existence of individual "preferences" for a particular type of error, it failed to reveal any more lawful general relationship between drug dosage levels and error fluctuations. Before the reasons for the comparative failure of the arithmetical task as a criterion

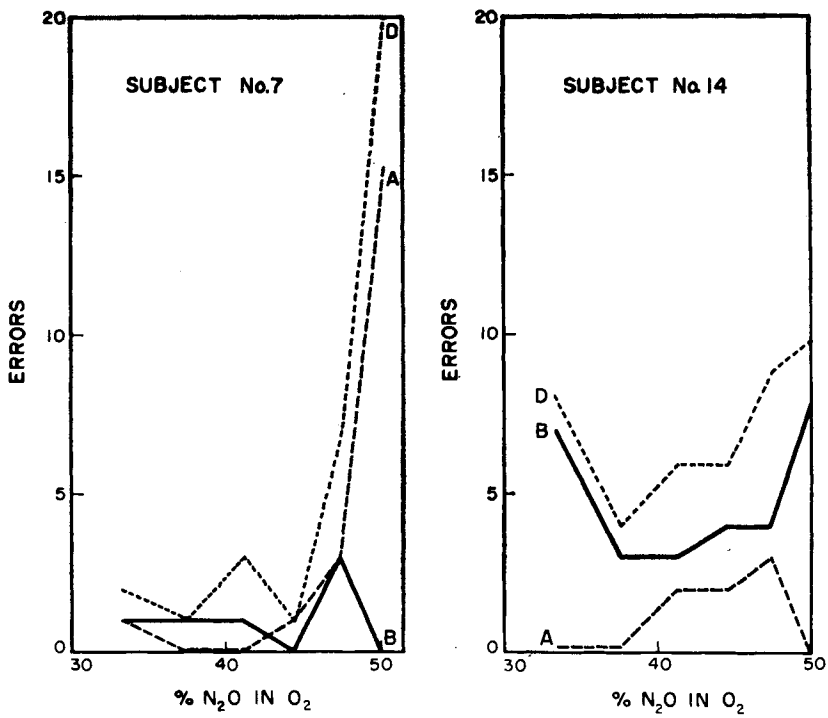


FIG. 6. Analysis of deterioration in performance of an arithmetical task under the influence of increasing concentrations of N^2O in O^2 for 2 subjects with contrasting patterns of error scores. (The scale of the graphs is one-half of the scale used in Fig. 5.) The total frequencies of non-responses (A), incorrect responses (B), and composite errors (D) are plotted. Perseverative errors have not been separately indicated; their contribution to the total composite error values in these 2 cases was minimal.

measure, and their implications, can be discussed, it will first be necessary to consider the results of other analyses carried out on the error scores.

Interrelationships between error categories — Correlations between the three error categories are reported in Table 4. These coefficients were

TABLE 4

Intercorrelations between Three Categories of Error Response in an Arithmetical Task

Sample Size	*Correlated Variables*	*r.*	*Significance*
25	$N-R \times W-R$	0·90	$p < 0{\cdot}001$
25	$N-R \times P-R$	0·29	N.S.
25	$P-R \times W-R$	0·54	$p < 0{\cdot}01$

calculated on the mean scores in each category over the entire range of gas concentrations. As the composite mean error at the penultimate "gas stage" was less than $12^1/_2$ per cent of the theoretically possible score and was still considerably less than 20 per cent at the final testing stage, it is possible that these correlation coefficients do not represent very stable interrelationships; at the 50 per cent level of nitrous oxide the pattern was tending to change towards a closer relationship between perseverative and wrong responses and a decreased correlation between the latter and non-responses.

TABLE 5

Correlations between Test and Retest Error Scores on an Arithmetical Task performed under the Influence of Various Concentrations of N_2O in O_2

	Sample Size	*Correlated Variables*	*r.*	*Significance*
	11	Mean Composite Error I × M.C.E. II	0·86	$p < 0{\cdot}001$
	11	$W-R$ I × $W-R$ II	0·85	$p < 0{\cdot}001$
a	11	$N-R \times N-R$ II	0·75	$p < 0{\cdot}01$
	11	$P-R \times P-R$ II	0·75	$0 < 0{\cdot}01$
	7	Composite error at 50% N_2O I × C.E. $_{(50\%)}$ II	0·76	$p < 0{\cdot}05$
b	7	$W-R_{(50\%)}$ I × $W-R_{(50\%)}$ II	0·27	N.S.
	7	$N-R_{(50\%)}$ I × $N-R_{(50\%)}$ II	0·75	$p = 0{\cdot}05$
	7	$P-R_{(50\%)}$ I × $P-R_{(50\%)}$ II	0·0	N.S.

Test-retest consistency — Eleven subjects were recalled for a second experimental session during which the same tests were performed under the influence of nitrous oxide administered under conditions identical with those in the original session. Nine of the 11 subjects performed the Pegboard and the Arithmetic tasks on both occasions; the other 2 were given only the latter task. Only 7 subjects received the entire range of drug dosages, 4 stopping one step short on either the original or the second occasion or on both. Correlation coefficients between first and second testings are reported in Table 5, (a) for the mean error scores over the total range of nitrous oxide concentrations, and (b) for the error scores obtained at the 50 per cent level of gas alone. Bearing in mind that retesting was carried out after periods ranging from a few days to almost a year, the intercorrelations of the mean scores in all error categories indicate a fair degree of consistency, although it is considerably lower than the "retest reliability" of the index of drug susceptibility derived from performance on the Pegboard test.

Relationship with personality variables — Product-moment correlation coefficients were computed between the mean composite error scores on the arithmetical task (excluding retest data) and measures of extraversion (E) and neuroticism (N). The personality scales were themselves uncorrelated (r EN = 0·03. n = 25), As in the case of motor decrement scores derived from Pegboard performance, the correlation with E was insignificant (r = 0·13); the relationship between the mean error scores and N was, however, significant though small in magnitude (r = 0·43 For 23 d.f. $p < 0·05$). When the total sample was subdivided into four personality groups as described on page 0.0 (E + N +, E + N −, E − N − and E − N +) the pattern of inter-correlations between error scores and E and N showed a similar configuration to that already reported between

TABLE 6

Intercorrelations between Performance Decrements on an Arithmetical Task and Two Personality Factors (Extraversion and Neuroticism)

Sample Size and Description	*Correlated Variables*	*r.*	*Stimulant*
Whole Group: n = 23	Mean Composite Error × E	0·13	N.S.
Whole Group: n = 23	M.C.E. × N	0·43	$p < 0·05$
E+Group: n = 11	M.C.E. × N	0·83	$p < 0·01$
E+Group: n = 11	Composite error at 50% N_2O × N	0·85	$p < 0·001$
E−Group: n = 12	M.C.E. × N	0·16	N.S.
E−Group: n = 12	$C.E._{(50\%)}$ × N	−0·40	N.S.
N+Group: n = 14	M.C.E. × E	0·41	N.S.
N+Group: n = 14	$C.E._{(50\%)}$ × E	0·30	N.S.
N−Group: n = 9	M.C.E. × E	−0·22	N.S.
N−Group: n = 9	$C.E._{(50\%)}$ × E	−0·62	$p < 0·05 < 01$

susceptibility to gas and personality. In the extraverted section of the sample, the relationship between neuroticism and error scores on the arithmetic task is positive and highly significant, and this holds equally true whether one considers mean error over the entire range of nitrous oxide concentrations or only the scores obtained at the 50 per cent level of gas. Table 6 contains the full set of coefficients obtained.

Relationship between gas susceptibility and error scores on the arithmetical task — The preceding data include no direct evidence that the deterioration in performance on the arithmetical task, as the concentrations of nitrous oxide were progressively increased, is due to the effects of the drug as such, rather than the result of repeated practice on the test or, indirectly, of the prolongation of a potentially anxiety-provoking test situation. Two methods were used to establish the effectiveness of the drug as an influence on test performance: (a) a comparison between performance under the drug condition and under placebo administration; and (b) the computation of correlations between the index of gas susceptibility (derived from motor task decrements) and error scores on the arithmetical task.

Placebo tests were carried out on 11 subjects, the interval between experimental and control tests ranging from 1 to 8 days. In 6 cases, the placebo condition preceded the drug test and followed it in the remaining 5. There were no discernable order effects. When the two sets of data were compared the following differences were noted: (1) The overall mean error score under placebo was significantly lower than under the drug condition ($t = 2{\cdot}51$: $p < 0{\cdot}05$); (2) The difference between the two sets of error scores at the sixth and final test trial was in the same direction and much larger ($t = 4{\cdot}97$: $p < 0{\cdot}001$); and (3) The distribution of error categories within the placebo test data differed from that in the drug data. No perseverative responses occurred under the placebo condition and the proportionate contributions to the total error of non-responses and incorrect responses were nearly equal (0·55 and 0·45 respectively); in the drug condition, non-responses accounted for 0·60 of the total error and incorrect responses for only 0·25. In summary, these data indicate that administration of nitrous oxide, in the range of concentrations used in this experiment, results in a highly significant deterioration in performance on the arithmetical task which is most marked at the highest dosage level and, moreover, that this depressant drug particularly encourages the commission of two types of error, namely perseverations and non-responses.

Further confirmation for the first of these conclusions is provided by a consideration of the relationship between error scores on the arithmetical task and the index of susceptibility to nitrous oxide. (The determination of this index is described on page 181) The correlation between the mean composite error on all test trials and the index of gas susceptibility is 0·52 (for 23 d.f., $p < 0{\cdot}01$); this rises to 0·62 when only the final test trial error scores are considered.

When the results of these various analyses of the arithmetical error scores are drawn together, the picture which emerges is that of a test involving a type of behavioural response relatively invulnerable to the de-

pressant effect of nitrous oxide, at least within the range of moderate dosage levels investigated. Since the task was chosen in the light of evidence indicating that it would, in fact, be more resistant to the effects of the test drug than would the finger dexterity task, this is to some extent merely a confirmation of expectation. We were wrong, however, in supposing that deterioration of motor performance at the highest gas levels might be too gross to allow of adequate discrimination between subjects with the lowest susceptibilities to the depressant drug, and wrong in our estimate of the degree of vulnerability of the arithmetic task. Both tests proved relatively less vulnerable than had been expected; in the case of the motor task, this was advantageous from the point of view of the experimental aims, but the opposite is true of the verbal task.

In the latter test, subjects started (with perfect scores) at a level of performance considerably below their potential maximum level. It seems reasonable to suggest that the behavioural effects of the depressant drug could, therefore, be counteracted for some time by an increased output of effort and that the random fluctuations in score within individual records over the first three or four drug dosage levels may, in fact, be a direct reflection of this process, perceived failures on test performance acting as the cue for heightened effort. The marked rise in the level of physical output which was observed to accompany responses to the verbal test in the last three stages of testing (e.g. raising of the voice, tapping of feet and clenching of hands) would fit in with such an explanation.

The psychological significance of the various types of error responses is unclear, particularly problematical being the instability or ephemerality of the perseverative response. This, although characteristically produced by certain individuals within a single test session involving the administration of gas, was never repeated by them (or anyone else in the sample) during placebo trials and tended not to be included in the same individual's repertoire of responses on occasions of retesting under nitrous oxide. If in addition to the previous facts, one also takes into account the comparative rarity of the perseverative response in test data, a plausible explanation suggests itself: this is that repetitions of the stimulus occur as responses when subjects want to respond, but momentarily forget what the task is (assuming that incorrect responses are more likely to occur when the task is remembered, but something goes wrong with the process of multiplication, whilst non-responses would result from inability or disinclination to attempt the task at all). Such an eventuality might follow as a reaction to failure stress or might index an involuntary rest pause (I.R.P.) from performance of what is, in effect, the only task component that is evoked by *all* stimulus numbers in common, irrespective of their separate identities. (Although there is a slightly higher proportionate occurrence of perseverative responses in the extraverted section of the sample than among the introverts, this is statistically quite insignificant.)

It may seem fanciful to suggest that subjects forget what they have to do with the stimulus numbers, but it should be remembered that during preliminary practice, the arithmetic and motor tasks were practised in separate unit trials, each trial being preceded by a brief recapitulation

of the original instructions. In the test session, on the other hand, the instructions were given once only, immediately preceding the 10 min period of oxygen inhalation. Subjects perhaps therefore had to acquire the habit of reminding themselves of the test instructions at the beginning of each successive presentation of the arithmetical task and, in view of the effects of nitrous oxide on habit acquisition (Steinberg and Summerfield, 1957) this habit may have been established only imperfectly. The uniformly decreased incidence of perseverative responses in individuals' retest data tends to support the suggested relationship with habit strength.

In view of their numerical insignificance, disproportionate space has been devoted to the examination of perseverative responses, the sole justification for which is the possible psychological significance of their occurrence only under the drug condition and not in placebo tests. If their investigation is to be extended, the first requirement would appear to be to devise some method of increasing the absolute frequency of their occurrence. If, as suggested, their appearance is related to an acceleration of the build-up of inhibitory potentials under the influence of a depressant drug, a promising first step would be to extend the length of each test trial beyond 2 min, or alternatively to increase the rate of stimulus presentation. The former alternative is, on the whole, to be preferred because: (a) a reasonable gap between stimuli is necessary to allow time for the subject to respond; and (b) an extension of the period of exposure to any given concentration of nitrous oxide would decrease the magnitude of error variance attributable to inter-individual differences in cerebral drug levels and thereby the validity of the behavioural measures.

SUMMARY OF RESULTS AND SUGGESTIONS FOR FUTURE RESEARCH

A method for the determination of individual differences in susceptibility to a depressant drug is described. The index of individual susceptibility is based on the mathematical function describing the course of deterioration in performance on a test of motor dexterity, with progressively increasing concentrations of nitrous oxide in oxygen. A second test which was administered at each level of drug concentration was one involving mathematical associative ability. Although this was less sensitive to the depressant effect of nitrous oxide than the motor test, measures of decrement from each test were significantly intercorrelated.

Performances on both tests under the influence of the nitrous oxide–oxygen mixtures were significantly worse than under the placebo condition where equivalent air–oxygen mixtures were administered. The index of drug susceptibility was found to be very stable on retests conducted after periods ranging up to a year from the date of the initial determinations.

A significant and complex relationship between susceptibility to the drug and the personality factors of extraversion and neuroticism was discovered and its implications discussed.

The most necessary extension of the present investigation now envisaged is to examine the effects of increasing the period of inhalation of the gas

concentrations to 10 min which, according to the work of Kety and Schmidt (1948a), should allow of a more complete stabilization of cerebral drug levels than it was possible to obtain in the present experiment. Repetition of tests at intervals within each 10 min period should indicate the extent to which our results have been influenced by possible individual differences in operative drug levels due to the unavoidable use of the shorter inhalation period, whilst the increased time available for testing would enable us to increase the duration of the arithmetic task as suggested above, as well as to investigate the possibility of significant individual differences in behavioural adaptation to the drug.

Chapter 7

THE EFFECTS OF MEPROBAMATE ON THE VISUAL AFTER-IMAGE

C. G. COSTELLO*

Introduction

In the writer's investigation of visual imagery, it was found, both that a depressant drug produced significant changes in some aspects of visual imagery (1957) and that differences between dysthymics and hysterics in the vividness and controllability of their imagery could be interpreted in terms of Eysenck's theory of cortical excitation–inhibition (1955). Further work on visual imagery was blocked by the problem of the strong element of subjectivity in studies of visual imagery. Deciding finally to transfer to more objective types of perceptual phenomena, it was found that experimental findings in relation to the visual after-image had been discussed in terms of Eysenck's excitation–inhibition theory (1957). For this reason the visual after-image was included among the perceptual tests chosen as the basis of further work on the effects of drugs on perception.

The investigation to be reported here had as its main aim the following: On the basis of the theory of excitation–inhibition, the drug postulate related to the theory and the data available on the after-image, to test predictions as to the effects of a depressant drug (meprobamate) on the duration of the after-image.

The above formulation of the aim of the investigation should not suggest that the investigation was designed to provide a rigorous test of the drug postulate; nor was it designed to provide conclusive evidence relating to the mechanisms underlying the effects of meprobamate; nor was it designed in the hope of providing a final explanation for the after-image. Forming the backcloth of this investi gation, we have the theory of excitation–inhibition and its drug postulate, the hypotheses relating to the action of meprobamate and the theories and hypotheses put forward to explain the after-image. None of these theories or hypotheses is strong enough to provide predictions the success or failure of which could be regarded as a crucial test of the other theories or hypotheses. Rather it was hoped that the findings obtained would, at one and the same time, throw some light on the adequacy of all these hypotheses and theories. The theory of excitation–inhibition did, however, play an important role as a starting point for thought and as a guide in the interpretation of the findings.

* The writer is indebted to the Wallace Laboratories for support.

Two subsidiary aims of the investigation were:

(1) To investigate the time course of the effects of meprobamate on the after-image.

(2) To investigate the difference in effects of a small dose (600 mg) and a large dose (1200 mg) of meprobamate.

The drug postulate of the theory of excitation–inhibition was given primary importance rather than the typological postulate of the theory. The Maudsley Personality Inventory (1959) was, however, administered to the subjects in order to obtain some data relating to the predictions as to the difference expected between extraverts and introverts on the after-image. Because the drug postulate was given primary importance in the investigation, it was necessary to do repeated testing of all the subjects over 3 days in the first experiment. This naturally reduced the number of subjects that could be tested and thus the typological postulate of the theory could not be adequately investigated. Even in the second experiment where the typological postulate was given greater importance, the number of subjects was only 20 and thus once again the experiment cannot be considered as providing an adequate test of the typological postulate.

Before embarking on the experimentation we asked ourselves the questions: (1) What is the evidence that the after-image is a central rather than a peripheral phenomenon? This is of the utmost importance since the theory of excitation–inhibition is a theory of central nervous system processes. (2) Can the data available on the after-image be predicted from the theory of excitation–inhibition?

Before presenting an account of the experiments, a review of the literature will be presented in an attempt to answer these two questions.

The definition of "central" always presents a problem, and necessitates a short discussion.

Eysenck (1957) writes "The term central... means anywhere within the central nervous system from a point separated by at least one synapse from the receptor organ on one side to a point separated by at least one synapse from the effector organ on the other."

At the present time, it seems to me that we must also further define central in terms of the particular function or phenomenon with which we are working. What is meant by central may change somewhat from function to function and indeed must do so, if it is to have an operational definition. Does this mean then that the concept "central", as used in Eysenck's theory of excitation–inhibition, is useless? It does not. Central, as used in the theory, is a short way of stating the proposition that differences between individuals in various perceptual and motor functions are due to basic constitutional differences between the individuals, and that these constitutional differences underlie more general personality differences. A peripheral theorist would say that the perceptual or motor function was constant from individual to individual, that inconstancies are due to poor conditions or to fluctuations in the subject's attention, co-operation, etc., and that personality differences rather than being related to important differences in the function merely produced slight differences of no importance, or differences that could be regarded as error of measurement.

The fact that Eysenck theorized about the nature of the processes involved should not lead one to think of "central" as implying definite localization.

One must then ask the central–peripheral question in relation to a specific function. The criteria of central as opposed to peripheral must be chosen for each function. It is only when we have done this for many functions that we can then ask: "Is the concept of "central", in the sense of localization, one that can be used generally for all functions, or must central mean something different for each function or group of functions, or is the concept "central" in the localization sense of any value at all?" It is only in this way, too, that we can hope to make the excitation–inhibition theory something more than a guide.

In asking the central–peripheral question in relation to specific functions, one finds that an even greater degree of specificity is required. One must relate the questions to specific aspects of the specific function! There seems to be little doubt, for instance, that photochemical processes play an important part in producing the complementary colours of the after-images of coloured stimuli. On the other hand, personality differences appear to be related to differences in the judgement of the size of after-images (Smith, 1957; Kragh, 1957). But in this investigation we are concerned with the conditions necessary for the occurrence of the after-image produced by an external stimulus, and the factors involved in the duration of such after-images.

In this review, we shall decide that central factors play an important part in determining the occurrence and duration of the visual after-image produced by an external stimulus:

(1) If we can show that stimuli presented to one eye have an effect on the occurrence and duration of the after-image produced in the other eye, or if after-images after binocular stimulation are different from those produced by monocular stimulation.

(2) If we can show that physiological stresses — drugs etc. — which have an effect on areas other than the retina produce a change in the occurence, and duration of the visual after-image.

(3) If changes in the occurrence, and duration of the after-image can be shown to be related to measurable cortical processes.

(4) If differences in the occurrence, and duration of the after-image can be shown to be related to measurable differences in personality.

Before presenting the evidence relating to the above four criteria, a word or two should be said about those studies concerned with after-images produced by visual images, dreams and hypnotic hallucinations. Oswald (1957) gives an account of reports by various workers of after-images resulting from the visual phenomena of dreams. He gives an account also of reports of after-images resulting from the coloured hallucination of hypnotic subjects and resulting from vivid visual images. Oswald himself reports that 15 of 20 subjects with vivid visual imagery experienced after-images of their visual images. Oswald concludes that after-images can occur as a purely central phenomenon. In this case, it should be noted that central refers to the absence of previous external stimulation. For this very reason we cannot claim

it as important evidence in relation to our problem since we are concerned with after-images produced by external stimulation. Oswald writes that: "The conformity of mental images with Emmert's Law demonstrates a cerebral correlate of angular size of a part of the whole visual field. This cerebral process persists in the absence of visual stimulation. The tentative conclusion might be drawn that the construction of a vivid image may involve many of the same neurones as are involved in the percept of a similar real stimulus". Only if this conclusion were shown to be valid, could we give much weight to the evidence in relation to our problem.

The same can be said with regard to studies, (Exner, 1888; Craik, 1940; Cibis and Northdurt, 1948; Oswald, 1957), showing that after-images may follow the exposure of a light source to an eye which is temporarily blind, the blindness being caused by local pressure on the eyeball. These studies, by functionally ruling out the cortex, suggest that retinal factors play a role in the production of after-images under normal conditions, in the same way that the studies mentioned above, by functionally ruling out the retina, suggest that cortical factors play a role in the production of after-images under normal conditions. But one must obviously beware of jumping too readily from these studies to conclusions about the production of after-images under normal conditions, and the studies referred to, though of some importance with regard to the occurrence of visual after-images, certainly cannot answer the question as to the importance of central and peripheral factors with regard to aspects of the normal after-image such as its duration.

Interaction Between Stimulation to Both Eyes

Let us look now at the evidence in relation to our first criterion. Consideration of the interaction between the eyes immediately reminds us of a similar problem — that of the transfer of the effects of stimulation in one eye to the other eye.

Parinaud (1882) was the first apparently to call attention to the fact that after-images could be seen if, after suitable stimulation to the retina of one eye, at the end of the fixation period, that eye be closed and the other (or resting eye) be directed to an evenly illuminated surface. Parinaud concluded that the seat of after-images is cerebral and not retinal. Bocci (1900) finds evidence in similar observations for supposing that the after-image seen by the "resting" eye (i.e. the eye which did not receive the primary stimulation or image) is of cerebral origin. Parinaud has been criticized (Delaberre, 1889; McDougall, 1904) on the ground of his failure to exclude the possibility that the after-image belonged to the eye on which the primary stimulus had fallen, and that such an after-image, which could be of retinal origin, was either combining or entering into rivalry with the field presented to the other (open) eye. Delaberre criticizes Binet who quotes from Parinaud in his "*Psychologie de Raisonnement*" the experiment on transfer described above, agreeing with Parinaud's conclusion. Delaberre points out the following facts: (1) the longer latency of the after-image of the "resting" eye;

(2) after obtaining the after-image, if one closes both eyes and then opens the resting eye, the image is dimmed or blotted out and is brightened when the resting eye is again shut. If the stimulated eye alone is opened, the opposite effect is experienced — when the eye is open the image is brighter, when shut it is dimmed; and (3) putting one's finger on front of the open stimulated eye results in the disappearance of the after-image, whereas putting one's finger in front of the resting eye produces no change. Delaberre comments that Binet and others have made the great mistake of supposing that "Whatever they might see in the field of the resting eye when it alone is open is actually seen by that eye". Creed and Harding (1930) found similarities between the after-image projected on to the black field of the closed stimulated eye and the after-image projected, supposedly, on an illuminated screen by the resting eye. They conclude that what is seen in the supposed transfer situation was the black projection after-image of the stimulated eye entering into rivalry with the white screen seen by the resting eye and temporarily displacing it in a marked degree from consciousness.

Sumner and Watts (1936) arrive at the same conclusion as Creed and Harding. Of particular interest in their experiments was the finding that Red, Blue and Black stimulus squares produced after-images in the transfer situation but that they were rarely produced by Green, Yellow and White squares. They suggest that the negative after-images of Green, Yellow and White cannot be distinguished against the black background of the closed eye and, therefore, are not so attention gaining as the white field seen by the open resting eye.

Ohwaki and Kihara (1953) have recently published some findings on the "So-called Bocci Image". They found: (1) only 6 out of 20 adults could produce the Bocci image;, (2) the latent time was long; (3) the Bocci image produced by a meaningful interesting stimulus was incomplete in its form and much simplified; (4) children of 7 to 12 years were able to produce the Bocci image. Only 50–60 per cent of children 13 to 14 years were able to produce it. The Bocci image appeared in only 14 per cent of the boys and 50 per cent of the girls in the group of 15 to 16 years; and (5) the duration of the Bocci image was remarkably longer than of the monocular after-image of the stimulated eye in the children of 7 to 12 years. These are the most important findings they list. They conclude that the Bocci image must be regarded as a characteristic special image which is to be distinguished from the binocular or monocular after-image. "The peripheral origin theory as represented by the contrast theory and the concurrence or fusion theory cannot explain the inability of so many adults to produce the Bocci image. It must be an image which is more strongly conditioned by central than by peripheral factors." It would be necessary to rule out the possibility of a difference between adults and children in the after-image of the stimulated eye against the black background of that eye when closed, and the possibility of differences in rivalry situations before we can accept the above conclusion. Though the findings on the whole are of interest, they do not support the conclusions of transfer. Indeed, some of their observations, such that some subjects saw the image clearly when they felt their eyes were tired

(relaxed accommodation?), and that the image disappeared when they wanted to see it clearly, would lend support to the rivalry or contrast theory. Hansen's findings (1954) that 15 out of 18 cases of subjects with anomolous retinal correspondence could "transfer" the after-image also supports the conclusion that the transferred after-image is not seen by the unstimulated eye.

It would seem that, at the present time, no investigator has successfully shown a true transfer of the after-image from the stimulated eye to the resting eye. Though the negative evidence in this respect cannot be used in an argument against the importance of central factors in the ordinary monocular and binocular after-images, the discussion has been presented in view of the absence to the writer's knowledge of a recent review of the problem.

Creed and Harding (1930) though finding no evidence for the transfer of after-images, did find: (1) binocular black projection images appeared earlier than right eye black projection images or left eye black projection images, and the binocular image was of longer duration. They conclude that uniocular processes reinforce one another (or are otherwise interacting); (2) the after-image transferred to the open resting eye appeared earlier than the black projection after-image of the closed stimulated eye. They conclude that the arrival of the transferred after-image is hastened by the formation of an image of the white screen in the resting eye and that this indicates interaction at a sub-perceptual level between the retino–cerebral apparatuses of the two eyes, and (3) that, after binocular stimulation, if one eye were closed, the open eye "saw" a black projection type of after-image with a shorter latency than monocular black projection images or binocular black projection images. This, they again interpret as evidence of interaction.

That there are limits to this interaction is suggested by Fry and Robertson's observation (1937) that if the retina to which the primary stimulus has been applied is faintly illuminated by letting light fall on the closed eyelid, a negative after-image appears, whereas it fails to appear if the opposite eye is similarly illuminated. It is quite possible that interaction will occur only above certain intensities. Certainly their observation does not justify the conclusion that in the case of fatigue images (images projected on a large bright after-field), fatigue in the synapses, if it plays a role, must occur in synapses below the convergence of the paths of the two eyes.

In keeping with Creed and Harding's findings with respect to the differences between binocular and monocular after-images, Misaik and Lozito (1951) found that binocular after-images had about a 15 per cent shorter latency and about a 24 per cent longer duration than the monocular after-images and interpret their findings in terms of interaction. Cooper (1948) also found that the mean image time of one eye only was shorter than of both eyes.

We may conclude that though there has been no successful demonstration of the transfer of after-images, the evidence in relation to the differences between monocular and binocular after-images satisfies our first criterion.

Effects of Stresses on Areas Other Than the Retina

Klein and Krech (1952) refer to an unpublished report by Klein to the effect that where persistence of the after-image was measured as a function of the duration of stimulus exposure, it appeared that, for longer exposures, the duration of the after-images in brain injured fell off significantly as compared with non-brain injured. Ruesch (1944) gave a battery of tests including a test of dark adaptation, a reaction time test and after-image test to 25 acute cases of brain damage, 32 chronic cases and 25 controls. The latency of the after-image for the three groups was, acute 13·3 sec; chronic 7·6 sec; control 7·1 sec. Ruesch also reports: "In order to analyse the association of the test results with persistent signs of brain damage following the initial loss of consciousness, the method described by Ruesch *et al.* was used to classify the cases according to the severity of the injury. In addition, the patients were separated according to the presence or absence of neurological signs. Only the results obtained with the negative after-image showed a significant separation of patients within the acute group. Those with neurological signs had a mean latency period of 16 sec and those without 8·4 sec.

Bock (1952) has reported that after-images are mainly affected in inflammatory, toxic or atrophic processes involving the path of conduction of visual sensation, while primary oedematous alterations have minimal influence. Diseases of the retina and choroid do not change after-images in a constant significant way. Similarly in glaucoma, after-images are usually normal so long as the nerve has not been damaged by pressure atrophy.

Bender and Teuber (1946) give an account of a patient with "areas of infraction on the under surface of both occipital lobes". The after-images of the patient, in defective areas, had a longer latency, were less vivid and of shorter duration.

Johnson, Bauer and Brown (1959) have reported that chronic brain syndrome patients showed more failures than normals on an after-image test.

Gellhorn and Spiesman (1935) found that with O_2 concentration 9–11 per cent, CO_2 concentration 4–7 per cent, or voluntary hyperpnea lasting for 2–5 min at a rate of 35 v and more per min, there is a lengthening of the latent period of negative after-images or even complete absence of any after-images. The effect may last as long as 12 min after the readmission of air. They conclude that their conditions produced "a lowered excitability of the fundamental nervous mechanism involved in vision."

McFarland *et al.* (1943) report that the latency of after-images in their study decreased as stimulus intensity increased — rapidly at first then more gradually. A given degree of anoxia caused a delay in the appearance of the after-image over the entire range of stimulus intensities, the magnitude of the delay being a function of the stimulus intensity. They also noted that, with administration of 100 per cent oxygen, the latent time did not recover immediately whereas the apparent dimming of the stimulus noted during anoxia recovered.

Though there may be some controversy over the location of the effects of oxygen lack, the evidence, on the whole, and particularly from the studies of brain damage, satisfies the second criterion.

Relation of After-Image to Cortical Activity

Travis and Hall (1938) found that for a relatively high degree of attention to the after-image, the total duration of *alpha* waves and the mean duration of the bursts during the after sensation periods were less, and the length of time for the first burst to appear after the light went off was greater than for relatively low degrees of attention.

Jasper and Cruikshank (1937) found that *alpha* rhythm was blocked during the presence of the after-image.

Popov (1956) reports "we have investigated the continued groups appearing on corticographical tracings taken from different cortical areas of the experimental animal (the rabbit) after various forms of stimulation (auditory, electrical, visual). By comparing them with the after-images seen by human subjects in EEG experiments, we have been led to identify the two phenomena."

There is also a report by Popov and Popov (1953) which more directly demonstrates the relationship between after-images produced by a sound as conditioned stimulus and the *alpha* rhythm of the EEG. The production, after ingestion of alcohol, of the longer latency of these after-images and sometimes their disappearance, along with the blocking of the *alpha* groups that were found in between appearances of the after-images before alcohol ingestion, can also be taken as further evidence satisfying the second criterion. It may be argued that, since Popov and Popov used after-images produced by a conditioned stimulus, we can give no more weight to the data than we did in the case of the after-images produced by visual images. Though there is an element of truth in this, the fact that the conditioned after-images were based on after-images produced by external stimulation makes the relationship a little closer.

Nagasaw and Obonai (1957) have also reported that after-images occurred with a decrease in *alpha* amplitude and their disappearance with an increase in amplitude.

We have then evidence satisfying criterion number three.

Relation of After-Image Differences to Personality Differences

There have been studies of relationships between personality differences and judgement of the size of the after-image, Kragh (1955), and Smith (1957). But there are only three studies that concern themselves with the duration of the after-image.

Brengelmann (1957) reported significant differences between normals, neurotics and psychotics in the duration of the after-image, normals having longer after-images.

Johnson, Bauer and Brown (1959) showed that schizophrenic patients performed poorly as compared with normals on the after-image test.

Levinson (1952) found significant positive correlations between the duration of the after-image and scores on the Hy and Pt scales of the M.P.I., the correlations being 0·53 and 0·59 respectively.

It is true that only the last mentioned study concerns itself with personality differences proper. The other two studies have been referred to here since it was felt that they were related more to this criterion than the others.

If we take only Levinson's study, we can say that the evidence, though scant, satisfies the criterion.

It may be that some would consider evidence showing the importance of any factors other than photochemistry in the production of the after-image as evidence in favour of the importance of central factors. For this reason a short account will be given of the evidence in relation to the part played by photochemical factors on the production of the after-image.

McDougall (1901) presents a number of observations which he considers as evidence that the fluctuations of after-images are not due to the cessation or reversal of action of chemical substances in the retina. (1) The after-images of two disks on either side of a fixation point disappeared alternately. (2) The period during which the after-image is capable of being revived is the same whether it is present to consciousness for the whole or only for a small part of the period. (3) He presents evidence showing that on very accurate fixation of a steady source of light and relaxation of all the muscles of the eye, both intrinsic and extrinsic, the image of the source of light might intermit, just as an after-image does. In this connection the findings of Crook (1930), Dunlap (1921), and Guilford (1927) are of interest. They show that there is a decrease in the sensitivity of a point on one retina when the corresponding part of the other retina has just been previously stimulated "thus showing", says Crook "the influence of central inhibition, central fatigue or some equivalent factor." (4) A direct image may be completely inhibited by another on the same retina. After a number of similar observations, McDougall writes: "I have reason to believe that the complete fading of visual images is due, not to a change in the retinal processes, but to a failure of the nervous impulses excited in the retina by the chemical changes to propogate themselves through those highest levels of the retino-cerebral system in which consciousness is immediately determined." Taking his observation as evidence against a purely photochemical process underlying after-images, we will consider his own theorizing later.

Judd (1951) refers to his own work (1927) on the complicated series of after-images yielded by a momentary flash of strong light which he considers as requiring an explanation involving more than photochemical changes.

Solomon and Werner (1952) produce some interesting findings on the importance of contour in the production of after-images. They demonstrated that while retinal stimulation remained the same, there were differences in perceived contour of a slowly rotating disk half black and half white. Either the white or black area was seen as a sector moving over the ground of the other area. The marked difference in after-image, according to the

perceived contour, once again can be considered as evidence against purely photochemical theories. In this connection Solomon and Werner also refer to Jablonski's finding (1930) that after-images from Ganzfeld stimulation are hard to elicit.

Creed and Granit (1928) present evidence suggesting that the latency of the after-image of disks as a whole is that corresponding to the region of retina on which the image of its edge falls. They interpret their results in other than photochemical terms.

McFarland *et al.* (1943) gives the following reasons why his data on the effects of anoxia on after-images cannot be explained in terms of photochemical mechanisms: (1) The light sensitivity of a previously dark adapted individual is reduced by subsequent oxygen deprivation. It is presumed that the concentration of photosensitive substances remains unchanged since the exposure to light has taken place. Furthermore the decrease sensitivity could not be due to delayed regeneration of these substances since this process had been completed before exposure to anoxia. (2) *In vitro* the rate of decomposition and regeneration of visual purple is not affected by the absence or presence of oxygen. (3) Although different photosensitive substances are concerned with rod and with cone vision, anoxia causes an equal rise in the rod and cone thresholds. (4) The threshold of the eye to electrical stimulation which presumably does not depend on photochemical mechanisms also rises during anoxia. (5) On readmission of oxygen, the rate at which sensitivity is recovered is much more rapid than can be accounted for by the rate at which the visual pigments regenerate.

Brindley (1959) has reported that, for the discrimination of the after-images of brief flashes, Weber's Law holds. At higher intensities, the Weber fraction $\Delta I/I$ increases very much. Brindley suggests that this increase is due to the bleaching of most of the visual pigment and not the saturation at some later stage in the transmission of visual information. He finds also that a given amount of light produces the same after-images (except for the first 15 sec) whether it is delivered within 15·7 msec or spread over 1·68 sec, and argues that this is consistent with the hypothesis that the after-image of a brief stimulus from the fifteenth sec until its disappearance 100–300 sec later, depends upon photochemical effects and not at all upon adaptation or potentiation of neural mechanisms on the retina or brain as a result of the intense activity that presumably occurs during and immediately after the stimulus. Brindley certainly seems to have demonstrated the importance of photochemical factors in this situation, but he himself comments that the inequality of the after-images in the first 15 sec is most reasonably explained by supposing that neural effects are then contributing. He comments also "Strong though I believe the evidence to be for the purely receptor origin of the after-images of brief stimuli, it does follow that neural factors are not concerned when the stimuli are long." In this review we are concerned mainly with the latter type of situation.

Creed (1931) on the basis of his findings relating to the after-image of a black disk rejects McDougall's (1901) photochemical explanation of the white after-image which results. McDougall's explanation is in terms of an X substance which diffuses into the black area from the illuminated

area during stimulation. Creed presents a mathematical type proof that no substances set free by photochemical action on the illuminated part of the retina can, under the condition of his experiment, have reached the middle of the unilluminated area.

The evidence available certainly in the case of stimuli of long duration and, to a certain extent, even for brief stimuli, clearly indicates that photochemical factors are not all important as they were once thought to be in the production of visual after-images.

We can now turn to the second question to be dealt with in this review: "How far can the data available on the after-image be predicted from Eysenck's theory of excitation–inhibition and its postulates?"

Eysenck (1960) has prepared a detailed theoretical formulation based on his general theory of excitation–inhibition in an attempt to mediate the relationships between the length of visual after-effects (including the after-image) on the one hand, and personality features, drug effects and brain damage on the other. He writes: "The experiment may be divided into two periods: (1) the period of *stimulation* during which the spiral or coloured surface is presented to the eye of the subject; and (2) after-effects or *reversal* period during which the subject experiences a subjective sensation. In what follows, I shall call the stimulation process X and the reversal $\bar{X}$; T refers to the time during which either process is in operation. During the period of stimulation *(T)* there is a constant increase in X; this may be assumed to take the form of a straight line function, although the actual slope and shape of this line are irrelevant to the argument. This theoretical function, however, is interfered with by a process of satiation which reduces the amount of X to a degree which is proportional to the satiability of the subject, i.e. it will be greater for extraverts than for introverts and for subjects having been administered a depressant drug as compared with subjects having been administered a stimulant drug." (It would follow also that in brain damaged subjects, the reduction of X would be greater than in non-brain damaged subjects). "The total amount of stimulation actually received therefore is a function not only of T, but also of the state of the organism."

"We now come to the reverse effect which is assumed to set in the moment the stimulus ceases to be effective, and which is assumed to be proportional in strength to the strength of the original stimulus effect i.e. $\bar{X} = X$. Because of the original satiation, X and consequently $\bar{X}$, will differ from person to person in terms of their degree of extraversion, drug administration etc. However, there is a further reason to predict that there will be a difference in $\bar{X}$ between these groups. It must be assumed that the process underlying the reversal is physiological in nature and consequently subject to satiation; this satiation will again be stronger in extraverts than in introverts, and in subjects having been administered a depressant drug as compared with subjects having been administered a stimulant drug" (and in the brain damaged as compared with the non-brain damaged), "these effects leading to a shorter after-effect period for extraverts and depressant drug subjects as compared with introverts and stimulant drug subjects" (and for brain damaged as compared with non-brain damaged), "are assumed to be cumulative; in any case the prediction is clear that after-effects will be shorter

for extraverts and depressant drug subjects" (and brain damaged subjects) "and longer for introverts and stimulant drug subjects" (and non-brain damaged subjects).

On the basis of this theoretical formulation then, one would predict the following:

(1) That extraverts would have shorter after-images than introverts.

(2) Depressant drug subjects would have shorter after-images than stimulant drug subjects.

(3) That brain damaged subjects would have shorter durations than non-brain damaged subjects.

(4) That successive stimulation by increasing satiation would result in a progressive shortening of the after-image. One would expect also, after a rest period, an increase in the duration due to the dissipation of satiation effects.

(5) That with increase in the duration of the stimulus, there would be an increase in the duration of the after-image due to the build up of X and that this would reach an asymptote marking the point when satiation prevented the further accumulation of X.

(6) Binocular stimulation by increasing X would produce longer after-image durations than monocular stimulation.

With regard to prediction (1), Nichols (1955) gives results in line with the prediction on after-image durations for 28 hysteric and 26 dysthymic patients. There was a tendency for the hysterics to experience the after-image over shorter periods than did the dysthymics the difference between means being significant on a one tail test of significance.

In the case of (2), Popov and Popov's finding (1953) that the after-image disappeared in some cases after ingestion of alcohol (a depressant) is in line with the prediction.

Klein and Krech's report (1952) of shorter duration of after-images in brain damaged than in non-brain damaged subjects is in line with prediction (3). Bender and Teuber (1946) report similar findings. Johnson, Bauer and Brown (1959) have reported that chronic brain syndrome patients showed more failures than normals on an after-image test, but unfortunately they do not provide data on after-image duration. Data on this aspect of the after-image are also lacking in Ruesch's (1944), and Bock's (1952) studies.

With regard to prediction (4), Klein and Krech (1952) found a decrease in after-image duration upon repeated exposure and that this decrease was more rapid in the brain injured than in the controls. Levinson (1952) gave his subjects 10 trials of an after-image test. He found that the mean duration of the after-image dropped sharply from the first to the second trial. The curve describing the decrease in duration was less steep from the second to third trial, reached a plateau from third to fifth trial and sloped asymptotically for the remaining trials.

Franz (1899), and Creed's finding (1931) that with increase in stimulation time, the negative after-image was more intense and of longer duration is in line with prediction (5). Berry and Imus's finding (1935) that as the log intensity X duration product increases arithmetically, the duration of the

after-image increases geometrically, is also in line with the first part of prediction (5). Nagamata's report (1951) that the duration of the after-image varied directly up to a limit of 80 sec is in line with both parts of prediction (5). Nichols (1955) also found an increase in the duration of the after-image with increase in stimulus exposure time from 5 sec 40 sec.

The increase in duration of the after-image following binocular stimulation as compared with monocular stimulation reported by Mizaik and Lozito (1951), Cooper (1948), and Creed and Harding (1930) is in keeping with prediction (6).

Where evidence is available then, it is in line with the predictions based on Eysenck's theoretical formulation.

How does Eysenck's theory of the after-image compare with other theories? Eysenck says nothing about the distinction between positive and negative after-images. To avoid a frequent confusion, "positive" and "negative" here refer to brightness and not to colour reversals. Theorists up to the present time have seemed to concern themselves exclusively with the distinction between positive and negative after-images — these theories were either unsuccessful, or could not be tested.

Typical of the theories concerning themselves solely with the difference between positive and negative after-images is that of Woodworth (1950). He mentions, first of all, Fechner's (1840), and Helmhotz's (1866) belief that there were two opposite after-effects of sensation. He goes on to describe Ebbecke's experiment showing the continual revival after stimulation of both positive and negative after-images by the changing of the projection field. The image becomes weaker and more evanescent. "Evidently", writes Woodworth "the after-effect is gradually dying out; and evidently both positive and negative after-images are indicators of the same after-effect." He describes another of Ebbecke's experiments (1929) showing that with a stimulus of moderate intensity fixated for a long time, he got a negative after-image on a dark field and not a positive after-image. "The reason for this difference," Woodworth comments, "is that with a stimulus field of moderate intensity fixated for a long time, local adaptation washes out the stimulus pattern and causes it to disappear even during the continuance of the stimulus as was vividly described by McDougall (1901)". (We have already noted similar results obtained by Crook, Dunlap and Guilford). "When this adaptation (retinal or cortical) has gone far enough the first after-image is negative on any after-field."

Ebbecke accounted for his results by a theory which assumes in the retina not two opposed after-effects but one effect only; this he regards as after-excitation. The adaptation which wipes out a stimulus pattern he displaces to the cortex or at least to some part of the brain. Woodworth rightly comments that the denial of retinal adaptation seems unreasonable in view of the difference between rod and cone adaptation. He comments also that there is a difficulty in the way of assuming enough retinal after-excitation to account for the after-images. "Prolonging the stimulus up to 60 sec or more increases the duration of the after-effect while the "maximum time" beyond which prolonging a stimulus does not increase the level of excitation and the brilliance of the primary sensation is only a fraction of a second."

Woodworth, having shown the inadequacy of Ebbecke's theory, proceeds to "modify" the theory, thus, producing one that is equally inadequate. He assumes that the single retinal after-effect is to be one of adaptation instead of excitation and by transposing to the cortex the necessary after-excitation to account for the positive after image.

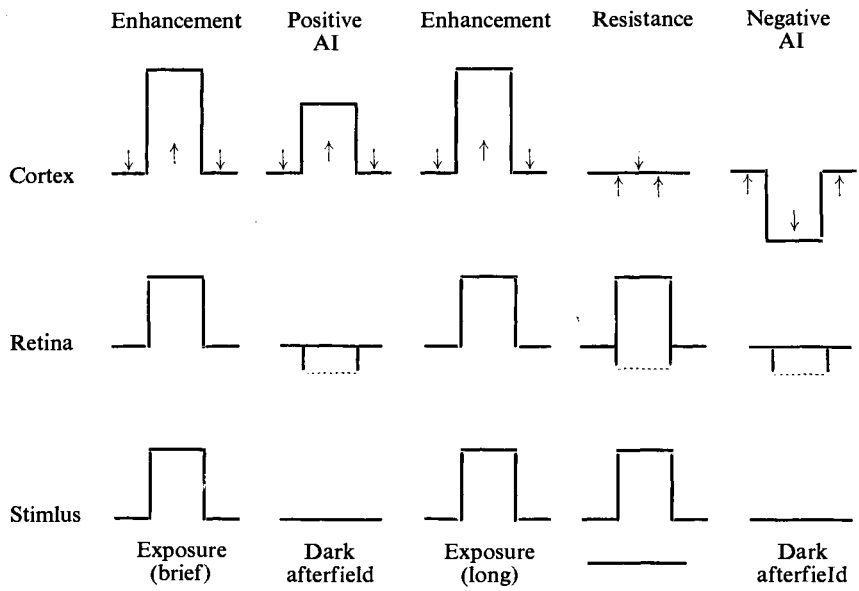


FIG. 1. (modified from Ebbecke 1929). A theory of positive and negative after-images seen on a dark afterfield. Up means brighter, down darker. The field is a bright spot with a dark surround. The retina is assumed to undergo adaptation (indicated by dotted lines) but no after excitation. The full lines opposite "Retina" indicate the pattern of stimulation sent up by the retina to the cortex. The cortex is assumed to take an active part in the proceedings (a) by enhancing or reinforcing a fresh pattern and (b) by resisting and neutralizing a persisting pattern of stimulation received from the retina. After a brief exposure the cortex is caught in the phase of enhancement and so gives a purely cortical positive after-image. During a longer exposure the cortex switches into the phase of resistance and so gives a negative after-image.

The above Fig. 1 is reproduced in its entirety from Woodworth. The first part of the figure is based on an over simplified picture of what happens after a brief stimulus. Judd (1937) gives the following sequence of events. (1) The primary image (not an after-image). (2) A very short dark interval (negative phase). (3) Positive after-image (bright) of same hue as primary image. (4) Dark interval. (5) Second positive after-image hue complementary to primary image. (6) Negative after-image. (7) Third positive after-image. (8) Final dark negative image.

Woodworth gives the above sequence of events himself later. It may be that in his figure he is concerned with different stimulus conditions. If so,

one would still require the above sequence to be explained, since his theory purports to be a theory of positive and negative after-images. It is fairly obvious that Woodworth's theory could not account for the above events. The second part of the figure concerned with the effects of a long exposure does not account for the fact that the negative after-image under such conditions may fade and return frequently. Indeed Woodworth's theory could not account for that fact. Similar criticism could be made against his attempt to explain the effects of alternately presenting dark and bright after-fields. Not only can these theories often be found to be inadequate to account for the facts with which they are concerned, they could usually be no more than plausible whereas a theory should be testable. The fault with such theories is that they present a too complex explanation of an oversimplified account of the facts, whereas the Eysenck type of theory presents a testable simplified theory as a first step in the understanding of a complex array of facts.

There is, of course, a quite different type of theory, or rather hypothesis, since it concerns itself with one finding in known conditions. For instance, Creed and Granit's finding (1928) referred to above that the latency of the after-image of disks as a whole is that corresponding to the region of the retina on which the image of its edge falls. In more detail, their finding that using small disks of constant size at varying distances from the fixation point, the latent period is longest in the centre of the field of vision, shortens rapidly to a point about 2° from the fixation point, then increases to a new maximum about 2° to 3° from fixation point and thereafter again shortens. The second maximum or 'hump' in the curve is interpreted as being due to the rods replacing the cones as the dominant receptor organs in and beyond this area. They consider the finding to afford evidence of the inhibition of cone mechanisms by the rod mechanisms. This kind of theory, hypothesis or explanation — whatever one wishes to call it — is excellent as far as it goes. It goes only as far as latency in connection with disks at certain distances from a fixation point and does not attempt to present a theory related to after-images under more general conditions. For that reason one cannot compare it with Eysenck's theory. Creed and Granit's approach should be considered as complementary to that of Eysenck's and at the same time their findings should be kept in mind when one is testing predictions from Eysenck's theory.

Review of Literature on Meprobamate

A word or two should be said here about the drug meprobamate in order mainly to justify our use of it as a depressant drug. Meprobamate (chemically: 2-methyl-2-n-propyl-1: 3 propanediol dicarbonate) sometimes known as Equanil or Miltown is described by its producers as "blocking internuncial neurones particularly in the thalamic region" and they state that it is indicated in "conditions requiring safe selective sedation or muscle relaxation."

With regard to the site of its action, Hendley, Lynes and Berger report that in a study of curarized cats, "The thalamus has shown consistent

changes in spontaneous electrical activity at lower dose levels than any other area. The increased amplitude and decreased frequency seen in this area are similar to the changes found in the cerebral cortex in association with decreased functional activity. Presumably, therefore, the thalamus is more or less specifically depressed by meprobamate. The thalamic areas affected include relay nuclei with direct cortical projections, such as nucleus ventrolateralis, nucleus ventroposteromedialis and the lateral geniculate nucleus, as well as n. centrum medianum which is part of the diffuse projection system (Jasper 1949) and probably projects to the cortex mainly through the basal ganglia (Gerebetzoff 1940). However, sensory relays through the thalamus are probably not greatly affected by meprobamate since no sensory impairment is apparent in animals, or reported by humans at normal dosage, and since auditory responses in our cat preparations are not affected except at very high dose levels (1957)." Their comment as to the effect in relation to sensory impairment is no longer tenable in view of the more recent findings to be reviewed below.

The specific thalamic effect of the drug itself has also been challenged recently. Pfeiffer (1957) presented evidence including studies of the effects of meprobamate on conditioned avoidance responses, strychnine and pentylenetetrazol threshold, spinal reflexes in animals, and on the EEG of humans, which led him to conclude that meprobamate "can be classified as a barbiturate-like drug with some CNS stimulant properties that may simulate those of trimethadione". Miletto, Colomb and Cardiare (1956) found that 60 mg/kg produced barbiturate-like generalized rapid rhythms. Tucker and Wilensky (1957) administered up to 4800 mg of meprobamate per day to chronic schizophrenic patients for 12 weeks and found increased fast activity in 18 of 32 subjects examined during the final week of medication. Henry and Obrist (1958) gave 2000 to 3200 mg of meprobamate by mouth in single doses and found fast wave increases usually after 30 min, in 10 of 12 patients. Shagass, Azima and Sangowicz (1959) administered to 12 psychiatric patients daily doses of meprobamate averaging 10 g for a period of 4 weeks. In all cases, EEG tracings looked like those obtained after intravenous barbiturates. The EEG sign of the sedation threshold which depends on measuring fast frequency activity was obliterated as amobarbital served only to augment slower frequencies than those usually measured. It was noted, however, that the behavioural depressant effects during meprobamate medication seemed to be less marked than might be expected from the EEG effects.

Though there seems to be some doubt now as to meprobamate's selective sedative effect, there is no doubt that it acts as a depressant. Further important evidence in this respect is presented by Eysenck and Eysenck (1960). Five tests — nonsense syllable learning, reaction times, level of skin resistance to the passage of an electric current, flicker fusion and perimeter threshold difference — were administered to 24 subjects under conditions of "no drug", Doriden and meprobamate medication. In a canonical variate analysis of the results, only one significant latent root is obtained showing that both meprobamate and Doriden are depressant drugs and do not differ from each other in their behavioural effects.

Meprobamate, although a depressant, does not appear to have such gross behavioural effects of sedation as barbiturates and is almost completely free from side effects. Marquis, Kelly, Gerard and Rapaport (1957) found no deleterious behavioural effects of 800 mg meprobamate in a study on motor skills, sensory processes and judgment necessary for safe driving. On the other hand, Reitan (1957) did find a significantly poorer performance as a result of administration of 1600 mg meprobamate when compared with a placebo using a battery of tests selected from Halstead's battery (1947). Reitan notes that adequate performance on these tests required altertness, sustained attention, accuracy of visuomotor coordination, motor speed, speed of reaction and problem solving. A second group which had received 400 mg meprobamate four times daily for 6 days preceding the testing, and 400 mg on the day of testing did not show a deterioration in performance when drug condition was compared with placebo condition. Rokeach, Oram and Marr (1959) found that neither analysis nor synthesis in thinking was affected for better or worse by 400 mg or 800 mg meprobamate in either high or low anxiety groups. They also reported that meprobamate did not seem to affect the subject's attitude toward the problem situation, his state of alertness or his memory functioning. Kornetsky (1958) using a multiple stimulus response apparatus found that 1600 mg meprobamate causes considerable motor impairment whereas 800 mg showed no effect. On the other hand, both 800 mg and 1600 mg produced impairment of learning stimulus response connections. More recently DiMascio and Rinkel (1960), and Klerman, DiMascio, Havens and Snell (1960) have reported that 800 mg meprobmate increased speed of mental activity (serial addition) and that there was no impairment in psychomotor functions (tapping speed, visuomotor coordination and steadiness). Drowsiness, reported with secobarbital and phenyltolaxamine, was seldom reported with 800 mg meprobamate. They found that 400 mg meprobamate improved psychomotor activity.

It would seem then that only in high doses does meprobamate have deleterious effects on visual and motor tasks. This is an advantage when working with sensitive functions such as the after-image. We will now go on to an account of the drug experiment using as our guide the prediction from Eysenck's drug postulate.

The drug postulate is — Stimulant drugs increase excitatory potential and decrease inhibitory potential, while depressant drugs decrease excitatory potential and increase inhibitory potential.

The prediction is — The duration of the after-image will be decreased by the administration of meprobamate — a depressant drug.

In a short preliminary study reported elsewhere (Costello 1960b), the significant results indicated: (1) that considerable differences existed between the subjects in the latency and total duration of the after-image; (2) that meprobamate, as predicted decreases the duration of the after-image; (3) that it also decreases the latency of the after-image; and (4) that there are differences between people in the effect of meprobamate on the total duration of the after-image.

Procedure

The stimulus conditions in this study were exactly the same as in the preliminary study.

The stimulus was a red square 5 cm^2 with a black focusing point in the centre. The red square, surrounded by black opaque paper, was attached to a sheet of perspex which was inserted into the end of the viewing tube of a Wither's tachistoscope (1954). Two 5 W neon lamps behind the perspex illuminated the red square during the stimulation period. All testing was done in a dark room.

After a number of preliminary trials to familiarize the subject with the after-image phenomenon, the subject was given a 3 min rest period. The subject was then given the following instructions: "In a few moments I shall ask you to look into the viewing tube. The red square will appear and I want you to focus your eyes on the black spot in the centre. After 30 sec the red square will disappear. As soon as the after-image appears, I want you to press the telegraph key on your right. As soon as the after-image disappears release the key. The after-image may appear and disappear a number of times. Remember — as long as the after-image is there, keep the key pressed down — as long as there is no after-image the key must be released."

The subject was then told to look into the viewing tube and the red square was illuminated. At the end of 30 sec, the illumination was switched off and a mark was made simultaneously on the recording chart of an Evershed and Vignoles recorder travelling at a speed of one inch every 5 sec. Activation of a second event marker by the telegraph key controlled by the subject recorded the appearances and disappearances of the after-image. When the after-image had completely disappeared, the subject was told to rest for 3 min and was then given a second trial.

Two scores were used: (1) the latency period, i.e. the time from the end of stimulation by the red square to the appearance of the first after-image, and (2) the total duration of the after-effect, i.e. the time from the appearance of the first after-image to the disappearance of the last after-image. It was decided to use this as the measure of duration rather than the time since the end of stimulation, due to the marked effects on the latency produced by the high drug dosage.

The analysis of variance in this study was done on the raw scores rather than on the means of the two scores at each session. This was done due to the large differences noted in some cases between the scores for the two trials. The recording method permitted accuracy to the nearest quarter of a second.

Each subject was given three treatments: (1) placebo; (2) 600 mg meprobamate; and (3) 1200 mg meprobamate on 3 different days, the order of treatments being counterbalanced for subjects and for sex. The tablets were in identically appearing tablet form and taken orally. The subjects were instructed to take a light breakfast and only water was allowed as a drink throughout the day.

On the first day of testing, the subject was seen at 9·0 a.m. Between 9 and 10 a.m., he was given practice on the spiral after effect test, the after-image test and the apparent movement test. During this session, initial readings were also taken on all three tests, the after-image being placed in time between the spiral and the apparent movement test. The drug or placebo was given at 10 a.m., the first post-drug session was at 10·30 a.m., lasting until 11·0 a.m., the second post-drug session was at 11·30 a.m. and so on throughout the day — half hour testing session, half hour rest period. In all, there were eight sessions; initial session, $^1/_2$, $1^1/_2$, $2^1/_2$, $3^1/_2$, $4^1/_2$, $5^1/_2$ and $6^1/_2$ hours after administration of treatment. A light lunch was taken between the second ($1^1/_2$ hours) and third ($2^1/_2$ hours) sessions after administration of treatment.

The weight of the subject was taken and the M.P.I. Eysenck (1959) was completed during one of the intervals between sessions.

The number of subjects in the experiment was 18 — 9 male and 9 female subjects. The ages of the sample ranged between 21 and 50 with a mean of 36. They were all paid volunteers of average intelligence or above.

Results

Table 1 shows the results of an analysis of variance carried out to test the significance of the differences between total duration scores for the two treatments.

TABLE 1

Analysis of Variance of After-Image Total Duration Scores

Source	*Degree of Freedom*	*S. Squares*	*Mean Squares*	*F*	*P*
Subjects	17	62272·36	3663·08	7·98	<0·001
Days	2	226·17	113·08	—	N.S.
Treatments	2	13886·12	6943·06	15·12	<0·001
Residual (1)	32	14689·82	459·06	5·02	<0·001
Total	53	91074·47			
Time	7	27089·28	3869·90	31·48	<0·001
Subject/Time	119	40256·54	338·29	2·74	<0·001
Day/Time	14	2201·54	157·25	1·28	N.S.
Treatment/Time	14	5867·83	419·13	3·41	<0·001
Residual (2)	224	27538·24	122·94	1·34	<0·01
Total	431	194027·90			
Final Residual (3)	432	39507·70	91·45		
Total	863	233535·60			
Between sex	1	1885·94	1885·94	—	N.S.
Between Subjects within sex.	16	60386·42	3774·15		

It will be seen from Table 1 that seven significant F ratios emerge, namely, those for "subjects", "treatments", "subject/treatment" interaction (Residual I) "time", "subject/time" interaction, "treatment/time" interaction and the "subject/treatment/time" interaction (Residual 2). Fig. 1 shows the results in diagrammatic form.

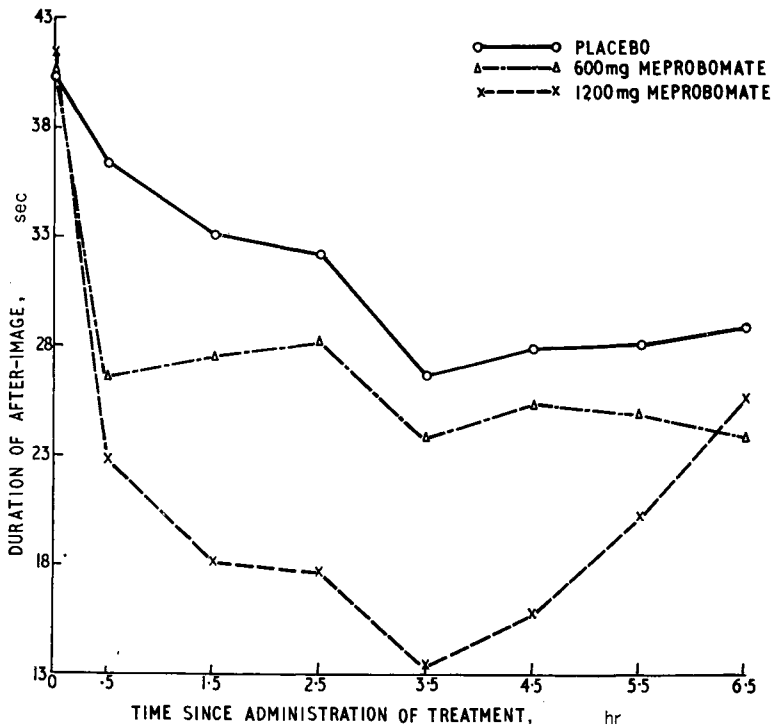


FIG. 1. Total duration of the visual after-image in seconds for the group of subjects under treatment in study II.

Neither the day of testing or the sex of the subject produced a significant degree of variance. The absence of a significant sex difference is in agreement with the findings of Morsh and Abbot (1945), and Pouliot and Misiak (1959).

Let us now look in more detail at the effects of the drug. A breakdown of the main treatment effect produces the following result:

	Mean duration
Placebo	31·72 sec
600 mg meprobamate	27·64 sec
1200 mg meprobamate	21·94 sec

Using the '*t*' test of significance we find that all the differences between the means are significant at the 0·01 level, and the results are in agreement with the results of the preliminary study (Costello 1960) and are in line with the prediction that meprobamate would decrease the duration of the after-image.

The significant "Subject/treatment" interaction indicates that the degree of the effect of meprobamate differs from subject to subject. Despite the small number of subjects, it was decided to correlate each subject's extraversion score with his percentage drop in duration of the after-image after administration of the drug.

It will be realized that there were numerous possibilities with regard to a measure of percentage drop in after-image scores since there were seven post-drug sessions for each drug. It was decided to take two measures for each drug level. For 600 mg, the two measures were: (1) the maximum percentage drop from the initial score for each subject on the 600 mg meprobamate day minus the maximum percentage drop on the placebo day. The maximum percentage drop on the placebo day was subtracted in order to get a measure of the drug effect alone. This score is known as AI.D.% decrease 600 mg (max.) — % decrease P. (max.) where AI.D. refers to after-image duration score; and (2) we also used the percentage drop for each subject at $1^1/_2$ hours after the administration of the drug minus the percentage drop $1^1/_2$ hours after administration of the placebo. This score will be known as AI.D. % decrease 600 mg (1—3) — % decrease P (1—3) where 1 in (1—3) refers to the first initial session and 3 to the third session $1^1/_2$ hours after administration of treatment.

Two similar scores were obtained as measures of the effect of 1200 mg of the drug.

It was felt that a measure at $1^1/_2$ hours after treatment and the maximum effect would give us measures of the beginning and maximum actions of the drug.

The correlations between these four scores of change and the extraversion scores are as follows:

	Extraversion
AI.D. %decrease 600 mg (Max.)—P(Max.)	−0·478 < 0·05
AI.D. %decrease 600 mg (1—3)—P(1—3)	−0·473 < 0·05
AI.D. %decrease 1200 mg (Max.)—P(Max.)	−0·380
AI.D. %decrease 1200 mg (1—3)—P(1—3)	−0·160

All the correlations are negative and two are significant at the 0·05 level. It will be remembered that Eysenck's theory predicted that extraverts should have shorter after-image durations than introverts — in other words there should be a negative correlation of an extraversion score with the duration of the after-image. The E score was correlated with the mean of the two duration scores for the initial session of each subject's first day of testing, the product moment correlation coefficient being

0·23, which, with 16 degrees of freedom, is not significant. No predictions were made with respect to the relationship between an individual's degree of extraversion and the degree of the drug effect, but if satiation builds up more readily in the extravert and our measures of change are measures of the satiating effect of meprobamate, then one would expect a positive correlation between E and the measures of change. The results are contrary to this prediction.

The first explanation of these results that suggested itself was that each individual reached a point of maximum satiation with the administration of a depressant drug and that extraverts reached this point more quickly than introverts (one perhaps can think here of the sedation threshold as used by Shagass) so that the drug in the case of introverts would continue to shorten the duration of the after-image after it had stopped doing so in the case of extraverts. Now if the initial score was, in actual fact, a measure of initial satiation (only indirectly so, of course, since the duration of the after-image is directly a measure of the continuation of excitation which has been reduced to a certain extent by satiation processes), then one would expect a significant positive correlation between the initial score and the amount of change. Unfortunately, we have already seen that there was an insignificant correlation between the initial score and E so that things are not promising. The actual correlations of our measures of change with the initial score are:

	Initial Score
AI.D. %decrease 600 mg (Max.) − P(Max.)	−0·34
AI.D. %decrease 600 mg (1 − 3) − P(1 − 3)	0·05
AI.D. %decrease 1200 mg (Max.) − P(Max.)	−0·34
AI.D. %decrease 1200 mg (1 − 3) − P(1 − 3)	0·38

The correlations are insignificant

The results of our correlations of drug effects with initial score are interesting but puzzling. They suggest that satiation as produced by a depressant drug is something different to that produced by stimulation with a red square and extraversion seems to be more related to the former. In this connection it is of interest that the test-retest reliability of the duration of the after-image based on the two initial scores is 0·89; based on the two scores $3^1/_2$ hours after administration of placebo, it is 0·77; $3^1/_2$ hours after 600 mg meprobamate, it is 0·60, and $3^1/_2$ hours after 1200 mg meprobamate, it is 0·34. This drop in reliability supports the suggestion of a difference in the satiation process. It would be of interest to see what effects of massed practice would be — one would predict on the basis of the above drug effects a negative correlation between extraversion and the drop in duration due to massed practice. Data in relation to this prediction are presented in the account of the second experiment.

A breakdown of the significant "treatment/time" effect produces the following results:

Session	*Means*		
	Placebo	*600 mg*	*1200 mg*
1	40·29 sec	40·60 sec	41·55 sec
2	36·48 sec	26·65 sec	22·88 sec
3	33·06 sec	27·53 sec	18·25 sec
4	31·75 sec	28·18 sec	17·73 sec
5	26·61 sec	23·94 sec	13·34 sec
6	27·92 sec	25·35 sec	15·78 sec
7	28·13 sec	24·95 sec	20·21 sec
8	28·94 sec	23·90 sec	20·77 sec

Applying the '*t*' test to the pairs of means the result is:

Session	*Placebo – 600 mg*	*Placebo – 1200 mg*	*600 mg—1200 mg*
1	N.S.	N.S.	N.S.
2	0·01	0·01	N.S.
3	0·05	0·01	0·01
4	N.S.	0·01	0·01
5	N.S.	0·01	0·01
6	N.S.	0·01	0·01
7	N.S.	0·01	N.S.
8	N.S.	N.S.	N.S.

From the above it can be seen that comparing placebo with 600 mg meprobamate, the drug has a significant effect at $^1/_2$ hour after administration and the effect remains up to $1^1/_2$ hours; at $2^1/_2$ hours it is no longer having an effect.

The effect of 1200 mg when compared with the placebo results is significant from $^1/_2$ hour to $5^1/_2$ hours after treatment; $6^1/_2$ hours after treatment it is no longer having an effect.

When we compare 600 mg with 1200 mg meprobamate, we see that a significant difference produced by the higher dose does not appear until $1^1/_2$ hours after the drug, that it remains up to $4^1/_2$ hours and then disappears.

We noted that there was a significant "subject/treatment/time" interaction suggesting that the effect of the drug on individuals occurred at different times. One would predict that the effect would occur earlier in extraverts than introverts since according to Eysenck's theory satiation

builds up more quickly in extraverts. In other words, one would predict a negative correlation between the time to maximum effect and E: for 600 mg meprobamate, r = 0·03; for 1200 mg, r = 0·08. Both correlations are, of course, insignificant.

One might predict that there would be a positive correlation between weight and time to maximum effect, i.e. the heavier the person the longer it would take for the drug to have its effect: with 600 mg; r = − 0·05; with 1200 mg, r = − 0·15. It may be argued that weight is not sufficient and that body build should be taken into account. This is probably quite true. In this study, however, numbers were too small to justify such elaborate measures. It is worth noting also that in practice it is rarely that body build is taken into account in the determining of drug dosage.

It was decided to correlate our four measures of change with weight with the following result:

	Weight
AI.D. %decrease 600 mg (Max.) − P(Max.)	−0·27
AI.D. %decrease 600 mg (1 − 3) − P(1 − 3)	0·35
AI.D. %decrease 1200 mg (Max.) − P(Max.)	0·33
AI.D. %decrease 1200 mg (1 − 3) − P(1 − 3)	0·08

With respect to the effects of meprobamate on the duration of the after-image then, we can tentatively conclude: (1) that meprobamate, as predicted, decreases the duration of the after-image and that 1200 mg produces a significantly greater effect than 600 mg; (2) that extraversion correlates negatively with the amount of change produced by the drug; (3) that weight is not significantly correlated with the amount of change produced by the drug; (4) that the subject's initial duration of after-image is not significantly correlated with the amount of change produced by the drug; (5) that both 600 mg and 1200 mg have an effect $^1/_2$ hour after administration of the drug, but that the effect of 600 mg lasts only until $1^1/_2$ hours after administration whereas the effect of 1200 mg remains up to $5^1/_2$ hours after administration; and (6) neither extraversion nor weight were significantly correlated with the time to maximum effect of the drugs.

All the subjects were also given the spiral after-effect test and apparent movement test. The results on these tests are reported in separate chapters. But a word should be said about the correlations between the scores on the after-image and on the other two tests. None of the correlations on the pre-drug scores were significant. Numerous correlations between the effects of the drug on the three tests were calculated. Only the correlation of 0·64 between the effects of the drug on the after-image and on the upper threshold of apparent movement $1^1/_2$ hours after administration of the drug was significant. We cannot place very much reliance on this one significant correlation out of many.

It is generally suggested that if cortical excitation–inhibition is the important physiological process underlying perceptual tests, then there should be positive correlations between scores on such tests. If the processes involved are as simple as postulated in the theory at present, this is

so. But the theory as formulated is a first step. It could not be hoped that things would be so simple, and that they are not so is obvious by the complex results that are being produced in experiments designed to test the theory.

It would seem clear that cortical inhibition cannot be considered a simple process produced in proportionate magnitude by apparently similar stimulating conditions. Nor can we be sure that a given amount of cortical inhibition will produce proportionate effects on measures of apparently similar perceptual after-effects and thresholds. We can only conclude, also, that though the predictions as to the effects of meprobamate on the after-image and the other two tests have been confirmed, thus substantiating the drug postulate that a depressant drug increases inhibitory potential, the magnitude of the effect of this increase of inhibitory potential on the tests used here is not proportional. This suggests a greater complexity than the theory as it stands indicates. One obvious possibility is a complex interaction between the inhibitory potential set up by the stimulation and the inhibitory potential produced by the drug.

This difference in the effect of the drug on the after-image when compared with its effect on the other two tests is revealed also when we consider the time course of the drug effects. Taking the 600 mg meprobamate first, we find that the drug has a significant effect on the after-image $^1/_2$ hour after administration of the drug and the effect remains only up to $1^1/_2$ hours after administration. In the case of the spiral, the "time/treatment" interaction was not significant, this being due probably to greater differences between subjects in the time course of the effect of the drug and also due to a greater persistence of the drug effect. The effect of 600 mg on the upper threshold of apparent movement descending trials is evident at $1^1/_2$ hours after administration of the drug and remains up to $5^1/_2$ hours after the drug. The effect on the ascending trials is evident $^1/_2$ an hour after the drug and remains up to $6^1/_2$ hours, no further testing being done beyond this time.

If we look at the effects of 1200 mg of meprobamate, we find that in the case of the after-image, the effect appears $^1/_2$ an hour after treatment, increases steadily up to $3^1/_2$ hours after treatment and then begins to disappear until it is no longer having a significant effect at $6^1/_2$ hours. The effect of 1200 mg on the spiral is similar to that of 600 mg, there being no significant "time/treatment" interaction. The effect of 1200 mg on the upper threshold of apparent movement (descending trials) is evident at $^1/_2$ hour after treatment and is still evident $6^1/_2$ hours after treatment when the testing ended. The effect of 1200 mg on apparent movement (ascending trials) is evident at $^1/_2$ hour after treatment and, though there appears to be a recovery from $4^1/_2$ hours, the drug is still having a significant effect at $6^1/_2$ hours.

The above differences in the time course of the effects of meprobamate on the three tests not only substantiate what was said above concerning the complexity of the inhibitory effect of the drug on the tests, but indicate the importance of determining for all functions the time course of drug effect.

Latency of After-Image

Table 2 shows the results of an analysis of variance carried out to test the significance of the differences between the latency scores for the two treatments. In the case of the latency scores, only the scores of 9 subjects could be used. In the case of the other 9 subjects, the after-image at one or more sessions on the drug days did not appear and there was no score available for latency. The 9 subjects used for the latency analysis were unevenly divided with regard to sex, there being 5 male and 4 female subjects, but, in view of the general non-significance of sex differences throughout this study, it is unlikely that anything was lost.

TABLE 2

Analysis of Variance of After-Image Latency Scores.

Source	*Degree of Freedom*	*S. Squares*	*Mean Squares*	*F*	*P*
Subjects	8	4632·09	579·01	59·51	<0·001
Days	2	28·16	14·08	1·45	N.S.
Treatments	2	37·71	18·85	1·94	N.S.
Residual (1)	14	136·24	9·73	2·10	<0·025
Total	26	4834·20			
Time	7	145·67	20·81	4·49	<0·001
Subject/time	56	545·19	9·73	2·10	<0·01
Day/time	14	160·68	11·48	2·48	<0·01
Treatment/time	14	112·71	8·05	1·45	N.S.
Residual (2)	99	548·06	5·53	1·19	N.S.
Total	216	6346·51			
Final Residual (3)	215	995·02	4·63		
Total	431	7341·53			

It will be seen that five significant F ratios emerge, namely, those for "subjects", "subject/treatment" interaction (Residual 1), "time", "subject/time" and "day/time" interaction. Fig. 2 shows the results in diagrammatic form.

In the preliminary study (Costello 1960) 600 mg meprobamate produced a decrease in the latency of the after-image significant at the 0·05 level. This has not been substantiated in this study and indeed Fig. 2 suggests a tendency for 1200 mg to increase the latency. In this study there is a significant "subject/treatment" interaction which was not found in the preliminary study.

In the report on the preliminary study (Costello, 1960), the significant decrease in latency produced by meprobamate was explained in terms of an inhibition by the drug of the positive processes preceding the negative after-image. This followed Levine and Graham's interpretation of their finding (1937) that as the intensity of an inducing patch increased the

latency of the after-image for a stimulus patch decreased. The results of this study suggest that this was a premature explanation based on inadequate evidence.

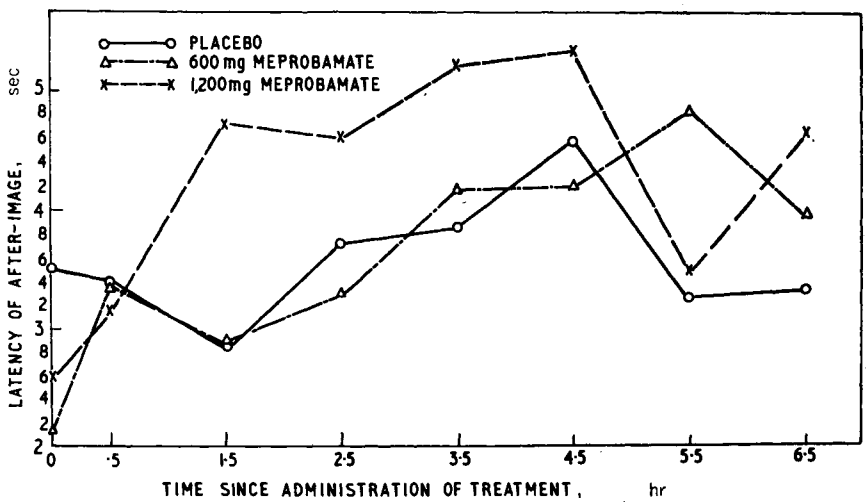


FIG. 2. Latency of the visual after-image for the group of subjects under drug treatment in Study II.

THE SECOND EXPERIMENT

A Comparison of the Effects of the Drug and of Massed Practice

Earlier, when discussing the negative correlations between the effect of meprobamate on the after-image and extraversion found in the first experiment, we wrote: "It would be of interest to see what the effects of massed trials would be. One would predict, on the bais of the above drug effects, a negative correlation between extraversion and the drop in duration due to massed trials".

It should be noted that Eysenck's theory, as it stands at present, would predict a positive correlation between extraversion and the drop in the duration of the after-image produced by massed trials. There is no evidence in relation to this.

One might also ask: Would the drop in duration of the after-image due to massed trials be greater after administration of a depressant drug than after administration of a placebo or vice versa? Eysenck's theory would predict a greater drop after the depressant drug. But the prediction based on the negative correlations between extraversion and the effects of meprobamate found in the previous study would be that there would be a greater drop after administration of a placebo.

The second experiment to be reported in this chapter was designed to test some of the above predictions. The predictions are many and will be listed when we consider the results of the experiment.

The total number of subjects in this study was 20. There were 12 male and 8 female subjects. All were paid volunteers. The ages of the sample ranged between 17 years and 47 years with a mean of 29·95 years.

The stimulus conditions for the after-image were the same as in the first study described above.

All the subjects were given a number of preliminary trials on the first day in order to familiarize them with the after-image and spiral after-effect phenomena. Two initial readings were then taken on the spiral test followed by two initial readings on the after-image test. The instructions, procedure, recording and scoring were exactly as in the first study.

The subject was then given the treatment. Each subject was given two treatments: (1) Placebo, (2) 800 mg meprobamate, on 2 different days, the order of treatments being counterbalanced. The form of administration of the treatment and instructions with regard to meals were the same as in the main study.

One and a half hours after administration of the treatment, the subject was given the spiral after-effect test. Six massed trials were given, each trial following immediately after the cessation of the after-effect produced by the stimulation of the previous trial. There was a 15 min rest period during which an auditory time-estimation test was given to the subject. This was followed by four more massed trials on the spiral.

After a 3 min rest period in complete darkness, six massed trials on the after-image test were given. Each trial followed immediately after the cessation of the after-image produced by the stimulation of the previous trial. This was followed by a 15 min rest period during which the subject was given a kinesthetic figural after-effect test. Four more massed trials on the after-image test were then given and the experiment for that day was complete.

During the $1^1/_2$ hour period after administration of the treatment of the first day, the subject completed the M.P.I. Eysenck (1959). Fig. 3 shows the results in diagrammatic form. Now we will consider the results in relation to the predictions.

(1) It is predicted, on the basis of Eysenck's theory, that the duration of the after-image will be decreased by the administration of meprobamate. It will be seen from Fig. 3 that the results are in line with the prediction, though the mean duration on the first post-treatment trial for the placebo day, 32·67 sec, is not significantly different from the mean duration of the post first post-treatment trial on the drug day, 28·59 sec.

This may be due to the time of testing. It will be remembered that in the first study there was a significant effect of 600 mg meprobamate at $1^1/_2$ hours after administration of treatment, but the difference was just significant at the 0·05 level. In this study we are not so concerned with the confirmation of the general drug effects found in the first study but in the subject/treatment interaction. It is for this

reason that the time interval $1^1/_2$ hours after treatment was chosen. Table 3 shows an analysis of variance done on the after-image data of this study. It will be seen that the main treatment effect is not significant. This is due to the significant "subject/treatment" interaction (residual 1).

TABLE 3

Analysis of Variance of After-Image Scores

Source	*Degree of Freedom*	*S. Squares*	*Mean Square*	*F*	*P*
Subjects	19	48406·22	2547·70	7·501	<0·001
Days	1	2·49	2·49	—	N.S.
Treatments	1	693·49	693·49	2·042	N.S.
Residual (1)	18	6113·44	339·63	4·690	<0·001
Total	39	55215·64			
Time	10	16280·56	1628·06	22·490	<0·001
Subject/Time	190	17428·26	91·73	1·267	<0·001
Day/Time	10	765·74	76·57	1·058	N.S.
Treatment/Time	10	414·22	41·42	—	N.S.
Residual (2)	180	13029·68	72·39		
Total	439	103134·10			

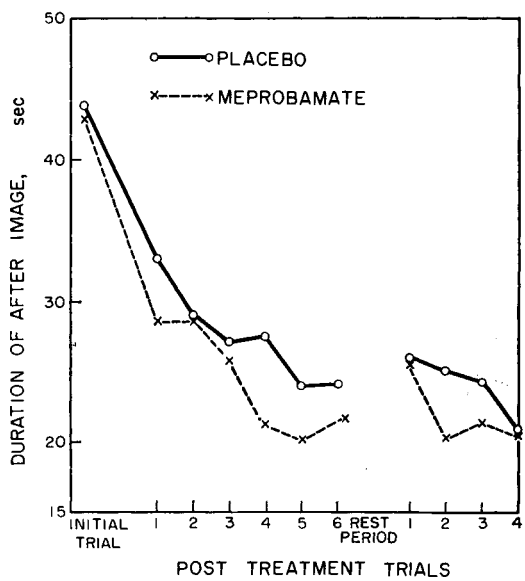


FIG. 3. The duration of the after-image for the group of subjects under drug treatment and conditions of massed trials.

(2) It is predicted, on the basis of the results of the main study, that there will be a negative correlation between extraversion and the percentage drop in the duration of the after-image produced by administration. of meprobamate. The correlation is 0·28. Here we have a correlation in the opposite direction to that predicted. We can only suggest the possibility that the session $^1/_2$ hour after administration of the treatment in the previous study is the crucial difference between the two experiments.

(3) It is predicted, on the basis of Eysenck's theory, that massed trials will produce a decrease in the duration of the after-image. Looking at Fig. 3 we see that the results are in line with the prediction. The decrease in duration from trial 1, 32·76 sec, to trial 6, 24·25 sec on the placebo day is significant at the 0·01 level (one tail). The decrease from 28·59 sec, to 21·61 sec, on the drug day is significant at the 0·05 level (one tail).

(4) It is predicted, on the basis of the findings of the previous study, that there be a negative correlation between extraversion and the decrease in duration of the after-image produced by massed trials. The correlation between E and the per cent drop from trial 1 to trial 6 on the placebo day is 0·15. On the drug day it is −0·17. Both, of course, are insignificant.

(5) It is predicted, on the basis of Eysenck's theory, that the drop from trial 1 to trial 6 will be greater on the drug day than on the placebo day. The drop on the placebo day is 15·65 per cent and on the drug day it is 25·8 per cent. The difference between these percentages is insignificant but the results are in line with the prediction. These results are not what would be predicted on the basis of the findings of the previous study. This is so despite the small positive correlation of 0·22 between the drop in the duration of the after-image produced by the drug and the drop from trial 1 to 6 on the placebo day.

(6) It is predicted, on the basis of Eysenck's theory, that after the 15 min rest interval, there will be an increase in the duration of the after-image due to the dissipation of satiation. Fig. 12 indicates that the results tend to be in line with the prediction. The increase from 24·25 sec on trial 6 (pre-rest) to 26·06 sec on trial 1 (post-rest) on the placebo day is not significant. The increase from 21·61 sec to 25·89 sec on the drug day is significant at the 0·05 level (one tail).

It would also be predicted, on the basis of Eysenck's theory, that there would be a greater increase in duration of the after-image after the rest period on the drug day than on the placebo day. The increase on the drug day is 65·4 per cent and on the placebo day, 19·8 per cent. Though the difference between the means does not quite reach a significant level, it is in the direction predicted.

Eysenck's theory would also predict a positive correlation between extraversion and the increase in duration of the after-image after the rest period. The correlation on the placebo day is 0·33 and on the drug day 0·12. Both are insignificant but in the right direction.

SUMMARY AND CONCLUSIONS

(1) A review of the literature on the after-image indicated that the after-image has important central components and that the data available was in line with the predictions from Eysenck's theory that:

(a) The after-images of hysterics would be shorter than those of dysthymics. Significant correlations between extraversion and initial after-image durations were not obtained in the present experiments. This is to be expected in view of the smallness and relative homogeneity of the groups used.

(b) That a depressant drug would reduce the duration of the after-image. This has been confirmed in the first study reported here. It was absent in the second study due to a significant "subject/treatment" interaction at the time after administration chosen for testing.

(c) That the after-images of the brain damaged would be shorter than those of normals.

(d) That successive stimulation would result in a decrease in the duration of the after-image. This has been confirmed in the second study reported here. The reminiscence effect after a period of rest, also predicted by Eysenck's theory, was found in the second study. It was also found that the decrease and reminiscence effect was greater after administration of a depressant drug. This result, though not quite significant, is also in line with Eysenck's theory.

(e) That with an increase in stimulation there would be an increase in duration of the after-effect.

(f) That binocular stimulation would produce longer after-images than monocular stimulation.

(2) The data on the after-image, on the whole, fit in very well with predictions from Eysenck's theory. Only the negative correlation found between extraversion and the effect of meprobamate in the first study is not in line with the theory as presented by Eysenck. If this is a reliable finding, a modification of Eysenck's theory to account for the data on the spiral after-effect, which is discussed in the spiral chapter and elsewhere (Costello, 1960e) may account for this one anomolous result on the after-image.

(3) It was not possible to make a prediction from Eysenck's theory as to the effects of meprobamate on the latency of the after-image. In a pilot study there was a slightly significant decrease in the latency produced by the drug. In the main study there was no statistically significant drug effect, but 1200 mg tended to increase the latency.

Chapter 8

THE EFFECTS OF MEPROBAMATE ON THE SPIRAL AFTER-EFFECT

C. G. COSTELLO*

The general framework of the author's drug experiments has been described in the chapter on the after-image and will not be repeated here. A review of the literature on meprobamate can also be found in the after-image chapter.

Before giving an account of the experiments, a review of the literature on the spiral after-effect will be presented.

Review of the Literature

We shall concern ourselves with two questions: (1) what is the evidence that the after-effect is a central rather than a peripheral phenomenon; and (2) how far can the data available on the test be predicted from the theory of excitation–inhibition?

The remarks made in the review of the after-image with regard to the general central–peripheral problem apply also to the spiral and will not be repeated. We shall also use the same four criteria in deciding whether or not the after-effect is a central rather than a peripheral phenomenon.

We shall, in other words, decide that central factors play an important part in determining the duration of the spiral after-effect:

(1) If we can show that stimulation presented to one eye has an effect on the duration of the after-effect produced by stimulation to the other eye, or if after-effects produced by binocular stimulation are different to those produced by monocular stimulation.

(2) If we can show that physiological stresses — drugs etc. — which have an effect on areas other than the retina produce a change in the duration of the after-effect.

(3) If changes in the duration of the after-effect can be shown to be related to measurable cortical processes.

(4) If differences in the duration of the after-effect can be shown to be related to measurable differences in personality.

Interaction between Stimulation to Both Eyes

We shall concern ourselves first of all with the evidence for the transfer of the after-effect from the stimulated eye to the resting eye.

Dvorak (1870), though he was concerned mainly to demonstrate the inadequacy of the eye movement theory of the after-effect, mentioned

* The writer is indebted to the Wallace Laboratories for support.

that he was able to transfer the effect from one eye to the other. In 1884, Budde reported an inability to transfer the after-effect. In 1888, Exner reported that he could get a transfer and in 1905, Szily reported that he could get it as did Ehrenstein in 1925. The next large group of workers who were concerned with the diagnostic value of the after-effect (see below) did not concern themselves with this question, though Price and Deabler (1955) did mention that they found the transfer of the effect in several of their normal subjects, but the most recent investigators, who are once more investigating the parameters of the effect, have concerned themselves with the question of transfer. Walls (1953) discusses the problem of transfer in relation to the visual after-image. He stresses the difficulty presented by the fact that the after-image can be seen by the closed eye and the fact that the so called after-image of the resting eye is actually the after-image of the closed stimulated eye. What is needed, he says, is an after-effect of such a character that it is seen only in the presence of, or as affecting the appearance of, a new visual stimulus, an after-effect, in other words, that cannot be seen by the closed eye. He assumed that the spiral after-effect cannot be seen by the closed eye. But as early as 1884, Budde reported that it could be seen by the closed eye and Stern reported similarly in 1894. Walls goes on to state that "a motion after-image is seen whenever the inducing and eliciting hemiretinas are correspondent but no effect is obtained when they are both nasal or both temporal hemiretinas." Since it is not something that is being seen with the closed eye that is subjectionalized within the field of the open eye, he argues that "information arriving in one cerebral hemisphere from one hemiretina can alter the state of the pathways which bring to that same hemisphere information from the homonymous hemiretina in the other eye. The transfer effect is intracortical, perhaps right in area 17, and is confined to one hemisphere. There can be no transfer of activity from the representation of one hemiretina in one occipital lobe to the cortical representation of either of the hemiretinas which project to the opposite occipital lobe."

Holland (1957) reports that there is a transfer effect of only 70 per cent. This suggests, he says, that there is a strong retinal element. This implies that he considers the transfer to be evidence for a cortical element in the after-effect. Pickersgill and Jeeves (1958) report that only 9 of 25 of their subjects were able to get the transfer effect.

Day (1958) criticizes Walls' argument and demonstrates that the transfer effect can be explained in terms of overlapping visual fields. He demonstrates also that the absence of transfer from one hemiretina to the hemiretina of the other eye projecting to the opposite hemisphere rather than being evidence in favour of cortical transfer in the case of interocular transfer within one hemisphere followed from the fact the fields do not overlap. The field of the stimulated part of the retina is now on a plain background. It might be felt that an after-effect of movement should then be seen on the white background since the stimulus (spiral, etc.) is certainly not necessary for the appearance of the after-effect. Yet, though Grindley and Wilkinson (1953) found that 19 of their 20 subjects saw the after-effect on a white field, Griffith and Spitz (1959) indicate the

necessity of texture for perceiving the after-effect. In the case of fusion of two fields, it is probable that quite definite texture would be required for the after-effect to be seen.

Day also quotes from a thesis by Seagrim who showed that for interocular transfer within one hemisphere, intensity was 50 per cent of maximum and that intensities for intraocular transfer and interocular transfer between hemispheres was 16·7 and 8·8 per cent respectively. The last percentage, though small, suggests that Walls' statement that "a motion after-image is seen whenever the inducing and eliciting hemiretinas are correspondent but no effect is obtained when they are both nasal or both temporal hemiretinas" is not altogether true.

As with the after-image then, demonstration of transfer or lack of it cannot provide us with any evidence for or against the central or peripheral nature of the after-effect.

Exner's (1888) report that the after-effect in one eye was diminished by stimulating the other eye by the opposite movement could be interpreted in terms of interaction or fusion of visual fields. Should the intensity of the after-effect only be affected, then it could be explained in terms of fusion. Should it be shown that the duration is affected, then it is more likely a case of interaction. On the other hand, it may be argued that fusion would simply produce a product of the two movements (perhaps an oscillation) or simply a rivalry of the two movements. Certainly Szily's report (1905) that stimulation by two opposing movements, one in each eye, results in an absence of an after-effect, if it could be repeated, could hardly be explained simply in terms of fusion of visual fields. Although this, no more than his observation that if one eye is closed after stimulation by opposite movement, one to each eye, the other eye sees an after-effect but a less intense one, cannot be considered strong evidence in favour of interaction. There is not then any good evidence to satisfy this criterion. On the other hand, there is certainly no negative evidence. Further work on monocular and binocular stimulation will need to be done perhaps with regard to the latency of the after-effect where fusion cannot be playing a role, before we can answer the question as to whether there are interaction processes involved in the production of the spiral after-effect.

Effects of Stresses on Areas other than the Retina

Much of the earlier work on the effects of brain damage on the spiral after-effect suffered from the crudeness of the all-or-none scoring technique. Nevertheless, the evidence does indicate that brain damage has an effect.

Price and Deabler (1955) tested 40 normals, 40 non-organic psychotics and 120 brain damaged subjects giving four separate tests and simply determining whether or not an after-effect was perceived. The brain damaged group had the greatest difficulty in perceiving the after-effect. A chi-square test of significance indicated that the brain damaged group was differentiated at the 0·001 level from the normal and functional groups.

Gallese (1956) tested 30 normals, 41 schizophrenics, 47 organics suffering from disorders other than those diagnosed alcoholic and convulsive

disorders, 50 organics suffering from alcoholic and convulsive disorders and 12 lobotomized schizophrenics. Gallese scores his test in terms of a cut between 2 or less and 3 or more reports of seen after-effects, calling the former an "organic" and the latter a "normal" score. A significantly greater number of organics had "organic" scores when compared with the normals or schizophrenics.

Garrett, Price and Deabler (1957) reported further findings on a group of 40 organics and 30 normals. Four trials were given. A normal report of the after-effect gained a score of 1, failure to perceive the after-effect scored 0. A report of forward or backward motion without change of dimension scored $^1/_2$. All of the normals obtained a score of 4. Only 2·5 per cent of the organic group were able to obtain a score of 4.

Page, Rakita, Kaplan and Smith (1957) tested 20 organics and 20 non-organic psychiatric patients, the two groups matched for age, education and length of hospitalization. Again significantly more organics than controls were unable to see the after-effect.

In Spivack and Levine's study (1957), 32 brain damaged boys and a control group of 35 non-organic boys "with primarily neurotic and characterological diagnoses" were tested. They report that 78 per cent of the brain damaged group perceived the after-effect on all trials (five trials were given) whereas 94 per cent of the control group perceived it on all five trials, a difference significant between the 2 per cent and 5 per cent levels of confidence. On the other hand, the mean duration in the brain damaged group was 20·2 sec and in the non-organic group 14·8 sec, a difference significant "at below the 1 per cent level of confidence by the median chi-square test".

Davids *et al.* (1957) tested 15 cerebral palsy children, 29 psychiatric children and 24 normal children. Using a scoring method with a maximum of 6, the results were;

	Mean score
Normals	5·5
Psychiatric	4·5
Organic	1·4

Holland and Beech (1958) tested 21 brain damaged and 17 normal controls. The organics were given four trials, the controls two trials. Only one patient out of the 21 organics failed to perceive the after-effect on all four trials. One other organic failed to perceive the after-effect on three out of the four trials, but otherwise, perfect scores were returned by other members of this group. No control subject failed to report the after-effect on each of the two trials given to this group. Though all-or-none scores did not discriminate very well between organics and non-organics in this study, a significant difference was found between the two groups on the duration of the perceived after-effect on trial 2: brain damaged mean duration: 11·29, controls 19·07. These results are, however, the opposite of those of Spivack and Levine (1957) who found that brain damaged had longer

durations of after-effect than non-brain damaged. We will refer to this once again when we discuss the predictions from Eysenck's theory.

Goldberger and Smith (1958) tested 30 normals, 17 non-organic psychiatric cases, 11 post ECT psychiatric cases and 24 organics. There were four trials and, using an all-or-none method of scoring the after-effect, a possible maximum score of 4.

The authors found a significant negative correlation between age and spiral scores, $r = 0{\cdot}39$. When comparing the three patient groups on the basis of their "means adjusted for age", they found no significant difference, but the organics did "maintain their low order position" among the groups.

Berger, Everson, Rutledge and Koskoff (1958) found that 31 of 110 patients in a neurosurgical ward has scores of 2 or less on the spiral using the Gallese scoring method. But they found that those who had score of 2 or less had less acute vision (as assessed by the Snellen cart) than the others.

Price, Garrett, Hardy and Hall (1958) in their further attempts to develop a test battery for the diagnosis of cortical brain impairment presented results on 50 chronic brain syndrome patients and 50 normals. They scored the four trials as in their previous study. They write, "In general, the results obtained in this study are consistent with the findings of other experiments and by other investigators. Though 90 per cent of the normal subjects earned a score of 4 on this test, only 0·06 per cent of the chronic brain syndrome subjects were able to achieve such a score. While 62 per cent of the chronic brain syndrome subjects failed to earn a score of greater than $^1/_2$, all the normal subjects were able to do so·"

Philbrick (1959) tested 81 patients in a neurosurgical ward. Agreement with regard to the diagnosis of 9 of these was not reached so that the final sample numbered 72. Of these, 45 were considered to be organic. Four spiral trials were given. She used the scoring method of Garrett, Price and Deabler (1957) and the cut off point of Gallese (1956) (0—2 organic, 3—4 non-organic). A chi-square test of her results was not significant. Philbrick comments that her results do not agree with those of other workers because she has used individuals falling closer to the centre of the distribution normal–organic. Though the sample is probably a more meaningful one for testing the efficacy of a clinical tool, the data produced by such a sample are not so damagingly negative with regard to our problem. This is as good a place as any to point out that I am not concerning myself here with the clinical value of the spiral test. In view of the numerous problems of type of brain damage, other variables such as extraversion–introversion, and the inadequately investigated effect of variables such as duration, size of spiral, expansion effect vs. contraction effect etc., it is not at all surprising that there is a great deal of conflicting evidence with regard to the clinical value of the spiral. For those concerned with the problem, the paper by Stilson, Gynther and Gertz (1957) on "Base rate and the archimedes spiral illusion" will be of interest and value. Gilberstadt, Schein, and Rosen's paper (1958) also deals with this problem.

Philbrick also tested 53 patients giving them the Weinstein Sodium Amytal test – Weinstein and Malitz (1954). The drug was administered until the patient showed signs of visual nystagmus, blurring of speech

drowsiness and counting errors (backwards from 100 by ones). Philbrick states that the Weinstein test "presumably increases certain organic symptoms when diffuse brain pathology exists." When scoring by the Price and Deabler method, i.e. using half scores, there is no difference between organics and non-organics. Using Gallese's method with no half scores, the difference between organics and non-organics was significant.

Johnson, Bauer and Brown (1959) tested 20 normals, 20 patients diagnosed "chronic brain syndrome" with indications of severe impairment (severe CBS), 20 patients diagnosed "chronic brain syndrome" with indications of mild impairment (mild CBS), 20 patients having a functional diagnosis and rated "severe" (severe functional) and 20 mild functionals. Four trials were given and the scoring method of Gallese used (1956), making a possible score of 4.

The authors write, "Performance of chronic brain syndrome patients was significantly inferior to that of normal and mild functionals on the spiral test ($p < 0{\cdot}01$). However, severe functional patients did not perform significantly better than mild or severe brain syndrome patients on the spiral test. And there was no significant difference between mild brain syndrome patients and severe brain syndrome patients on the spiral."

Although the results are not favourable with regard to the efficiency of the spiral test in differential diagnosis, they are in agreement with the criterion with which we are concerned.

Spivack and Levine (1959) tested 24 brain damaged subjects (BD) and 20 normals (C) with the spiral visual after-effect (SVA) test and other tests. Each subject was given: (1) 4 SVA trials at 30 sec exposure, (2) memory tasks; (3) 5 SVA trials at 5 selected exposure times; (4) necker cube; (5) repeat of 3 above; (6) figural after-effect test; (7) repeat of 3 above; (8) Schroeder staircase; (9) repeat of 3 above; and (10) delayed recall of memory tasks. The battery of tests was given once more 3 months later. The authors report that 11 of 24 BD subjects or 46 per cent failed to report SVA on one or more of the four intial 30 sec trials. All of the subjects reported SVA on all four 30 sec trials. On retest one month later, only 22 per cent of the BD subjects failed to report SVA on one or more of the trials, while the other C subjects continued to report the effect consistently.

Truss and Allen (1959) tested 17 cerebral palsied subjects (CP) and 8 normal subjects. The 17 CP subjects were split into two groups, the first experimental group ($N = 7$) being tested 3 months earlier than the main experiment group ($N = 10$). Subjects were given a randomized pattern of five 10 and five 30 sec exposures. The intra-subject variability in duration of the after-effect for 10 sec exposures was significantly greater for the organics than for the normals.

Going from the studies of brain damage to those of drug effects, we have first the Eysenck, Holland and Trouton study (1957). Six subjects were tested and were administered in counterbalanced order, in the form of a double Latin square, a placebo, 290 mg sodium amylobarbitone and 10 mg d-amphetamine sulphate. Four trials constituted the test procedure. It was found that Amytal significantly reduced the duration of the after-effect when compared with Dexedrine and placebo though Dexedrine did

not significantly increase the effect when compared with placebo. Eysenck and Easterbrook (1960) tested 8 subjects, the drugs being dextramphetamine sulphate, sodium amylobarbitone, meprobamate and a placebo. The mean duration of after-effects was 24·05 sec under placebo conditions, 29·64 sec under amphetamine, 23·11 sec under Amytal and 24·97 sec under meprobamate. The only significant difference was that between amphetamine and placebo.

Of the 14 studies of brain damage reviewed, we find that 11 present significant differences between organics and non-organics, 1 study presents non-significant differences which are, nevertheless, in the right direction, another study indicates some difficulty on the part of patients in a neurosurgical ward in seeing the after-effect but finds the difficulty is related to visual acuity and 1 study presented data indicating non-significant difference between organic and non-organic patients in a neurosurgical ward. As mentioned above, such non-significant differences might have been expected between such groups. But even in this study, a significant difference between the two groups was indicated after administration of Sodium Amytal. Both drug studies indicate that drugs have a significant effect on the spiral after-effect.

We can say then, that the evidence satisfies the criterion. The conflicting evidence with regard to the actual effect of brain damage on the duration will be discussed in relation to the prediction from Eysenck's theory.

Before proceeding to the next criterion we should mention the study by Freeman and Josey (1949) purporting to show a relationship between absence of the spiral after-effect and the degree, by clinical estimate, of the impairment of memory. Unfortunately, one of the authors informs me that the results reported may not be correct (Freeman 1959). Although Standlee's results (1953) failing to find a correlation between the after-effect and memory ability as measured by the Wechsler memory scale, should not, as Price and Deabler (1955) point out, be compared with the findings of Freeman and Josey since they were compared with memory impairment rather than memory ability, for the reason stated above, we have no reliable evidence on this matter at the moment.

Although not directly relevant to the criterion of brain damage, it is as well to mention here also the study by Harding, Glassman and Helz (1957). They tested 81 children ranging in age from 48 to 71 months. No children below 55 months reported the after-effect, while all those above were able to report the phenomenon. Mental age was found to bear a more direct relationship to perception of the after-effect than chronological age. The authors conclude that "the results of the study lend support to the hypothesis that children below a certain age level, presumably because of insufficient neural maturation, exhibit some behaviours similar to those of brain injured aduals."

But Gollin and Bradford (1958) have shown that 6 of 23 children tested for the spiral after-effect under a method designed to obtain their responses under actual as well as illusory conditions, failed to see the illusory after-effect. Five of the 6 subjects also failed in the situation where they had to respond to actual changes. Also important is the general finding that 17

of the 23 children could see the after-effect though they were younger than the subjects of Harding and his associates.

Noteworthy also is Aaronson's report (1958) that in his study, 65 epileptic patients' perception of the spiral after-effect was significantly related to both visual and auditory impairment and that performance on the spiral deteriorated as more sensory avenues became impaired. He found also that there was a strong tendency for the non-perception of the after-effect to be related to even mild degrees of aphasia.

Indirectly related to this criterion also is the study by Spitz and Lipman (1959). They report that in their investigation all of their 32 normals perceived the spiral after-effect whereas 9 of 41 retardates failed to see the after-effect.

Relation of the Spiral After-Effect to Cortical Activity

To the writer's knowledge there is no evidence relating to this criterion. Since one can apparently see the after-effect when both eyes are closed, there should be no difficulty in studying the relationship between the after-effect and the EEG.

Relation of the After-Effect Differences to Personality Differences

Eysenck, Holland and Trouton (1957) report that the 4 most introverted subjects (as measured by the M.P.I.) of 17 subjects tested showed longer spiral after-effects than the 4 most extraverted. Holland (1960) found that two measures of the duration of the after-effect correlated with two questionnaire measures of extraversion produced negative correlations between duration of after-effect and extraversion – three of them significant at the 5 per cent level.

On the other hand, Pickersgill and Jeeves (1959) found no difference in the duration of the after-effect between the 12 extreme extraverts and 12 extreme introverts of their 76 subjects.

But Lynn (1959) tested 40 subjects and found that the correlation between the duration of the spiral after-effect and E was $-0{\cdot}43$ ($p < 0{\cdot}05$).

Claridge (1960) gave four spiral trials to a group of 16 normals, 16 dysthymics, 16 hysterics and 16 schizophrenics with the following result:

	Spiral after-effect	
	Mean	S.D.
Normals	10·16 sec	4·560
Dysthymics	15·78 sec	5·486
Hysterics	9·74 sec	5·337
Schizophrenics	14·78 sec	6·996

The spiral after-effect of the hysterics was significantly shorter than that of the dysthymics ($p < 0{\cdot}01$), that of the dysthymics significantly longer than that of normals ($p < 0{\cdot}01$), that of schizophrenics significantly longer than

that of normals ($p < 0{\cdot}05$). Within the normal group, r between the E score of the M.P.I. and length of after-effect was $-0{\cdot}596$ ($p < 0{\cdot}01$).

Eysenck (1960) reports the factor analysis of a variety of tests and questionnaire scores. His Factor I he identifies with extraversion – the positive end of the Factor being the introverted end. Two measures of the spiral after-effect used in Holland's study (1960) referred to above had loadings of 0·726 and 0·685 on this factor.

We may conclude then, that criteria 2 and 4 have been substantially satisfied, and that there is some positive evidence in relation to criterion 1 – all of which suggests that the spiral after-effect has a strong central component.

We now turn to the second question to be dealt with in this review: "How far can the data available on the after-effect be predicted from the theory of excitation–inhibition?"

Eysenck's theoretical formulation as outlined in the Chapter for the after-image, being exactly the same for the spiral after-effect, it will not be repeated here. It may, however, be pointed out that Eysenck's more detailed theoretical formulation which takes into account both the inhibition set up by the original stimulation and that produced by the after-excitation, takes care of Storms and Sigal's criticism (1958). It also makes more clear how the predictions made in Eysenck's *Dynamics of Anxiety and Hysteria* (1957) follow from the theory. The predictions based on the theory are the same for the spiral after-effect as for the after-image.

(1) It is predicted that extraverts would have shorter after-effects than introverts. We have already seen that five reports, Eysenck *et al.* (1957), Holland (1960), Eysenck (1960), Claridge (1960), and Lynn (1959), provide evidence in line with the prediction. Only one report, that of Pickersgill and Jeeves (1959), presents data showing no difference between extraverts and introverts.

(2) It is predicted that depressant drug subjects would have shorter after-effects than stimulant drug subjects. The findings of Eysenck *et al.* (1957) that Amytal significantly reduced the duration of the after-effect are in line with the prediction as are Eysenck and Easterbrook's (1960) findings, that amphetamine increased the duration. Other findings in both studies are neutral with regard to the prediction.

(3) It is predicted that brain damaged subjects would have shorter durations than non-brain damaged subjects. Here the evidence is conflicting. Page, Rakita, Kaplan and Smith (1957) found no significant difference in the duration of the after-effect between their organic and non-organic groups. Unfortunately, they do not give data that would indicate in which direction the trend lay. Holland and Beech (1958) reported significantly shorter durations of the after-effect for their organic group, but Spivack and Levine (1957), Spivack and Levine (1959), and Truss and Allen (1959) found significantly longer durations for their brain damaged groups. Gallese (1956) reported his impression that organics had shorter durations than non-organics and Philbrick (1959) gave a similar report.

We have then, one objective and two subjective reports in favour of the prediction, three objective reports not in line with the prediction, the

data of the seventh report showing no significant differences. The results as they stand then, are contrary to the prediction from Eysenck's theory.

In trying to account for the differences in results, it was found that though the different investigators used a variety of exposure times, all included an exposure time of 30 sec and the above results held for this exposure time so that time of exposure or stimulation does not seem to account for the discrepancy. On the other hand, the results in line with the prediction were obtained from studies using both the expansion and contraction effect whereas the studies reporting findings contrary to the prediction used only the expansion effect. In view of the differences generally found between expansion effects and contraction effects (Costello 1960 d and e), it is thought that this difference in administration of the test may account in part for the difference in results obtained.

It may be argued that one should regard those studies reporting failure to see the after-effect in the case of the brain damaged to be in favour of Eysenck's theory. But perhaps those studies concerned with all-or-none effects should be handled with caution. London and Bryan (1960) have reported data on 44 brain damaged subjects and 22 normal controls. They found that organics given structured instructions (a description of possible after-effects) reported after-effects almost as frequently as did normals, while organics with neutral instructions were relatively unable to report the phenomenon. They interpret their results in terms of Goldstein's theory that the lives of organics are oriented towards the avoidance of "catastrophic reaction" and his theory of the inability of organics to deal with abstract situations.

The theory, as it stands, can only account for complete failure to see the after-effect on the part of the organic. It does not account for differences in the duration of after-effect between organics and normals. We may criticize also London and Bryan's unhappy attempt to link Goldstein's theory with Shapiro's (1954) theory in which he interprets the failure of organics to see apparent movement in terms of an "exaggeration of inhibitory effects" in brain damaged patients. The attempt results in a peculiar mixture of psychological and physiological concepts. The experimental results do however justify our caution.

(4) It is predicted that successive stimulation by increasing inhibition would result in a progressive shortening of the after-effect and that after a rest period, there would be an increase in duration (reminiscence) due to the dissipation of inhibitory effects.

Wohlgemuth (1911) reports that by alternating spirals (one giving an expansion effect and one giving a contraction effect) the after-effect is rapidly decreased over successive stimulation. With a stimulation time of 20 sec and 50 trials, the after-effect dropped from a duration of 16 sec to one of 4 sec. He notes that there was no further drop during the last 20 trials. Wohlgemuth interprets his findings in terms of the interactions between spirals of opposite sign. Although he mentions that there was a tendency for the duration to increase with repeated stimulation using the same spiral, he does not give any observations, and the possibility that

the decrease in duration with the two different spirals is due simply to a build-up of inhibition rather than a more complex interaction process, was not sufficiently investigated.

Eysenck, Holland and Trouton (1957) gave their subjects four trials of 60 sec stimulation with rest periods between trials of 60 sec. The means for the four trials were: 23·83 sec, 21·38, 21·16 and 20·27. The difference between the means was significant at the 5 per cent level.

Pickersgill and Jeeves (1958) report that there was a difference in duration of after-effect produced by same stimulation conditions at the beginning and at the end of a series of thirteen intervening measurements — the difference being significant at the 1 percent level.

Spivack and Levine (1959) gave their subjects a battery of tests in the following order: (1) Four SVA trials (spiral visual after-effect) at 30 sec exposure; (2) Memory tasks; (3) Five SVA trials at 5 selected exposure times (5, 10, 20, 30 and 50 sec); (4) Necker cube; (5) Repeat of 3 above; (6) Figural after-effect; (7) Repeat of 3 above; (8) Schroeder staircase; (9) Repeat of 3 above; and (10) Delayed recall of memory tasks. All the subjects were given the same battery one month later.

Comparing the mean response in the first block of five trials with the mean response obtained in the fourth or last block of five trials, 17 of 20 normal subjects showed a drop ($p < 0{\cdot}001$) and 13 of 20 brain damaged showed a drop ($p = 0{\cdot}10$). Comparing the sum total of all trials excluding the initial four 30 sec trials for the two session which were separated by a one month period, there was a significant drop in duration for both groups at the 0·01 level. This would seem to be due to something other than inhibition as used in Eysenck's theory.

Lynn (1959) presents data on 40 subjects confirming his prediction that "with repeated presentation, extraverts should show a greater fall off in the after-effect as a result of their tendency to accumulate reactive inhibition quickly ($p < 0{\cdot}05$). His data was also in line with the prediction that after a period of rest, extraverts should show a greater recovery in seeing the after-effect as a result of the dissipation of reactive inhibition, but the difference was not significant. This may have been due to the too short 2 min interval allowed for the dissipation of inhibition.

London and Bryan (1960) noted that in their study normal and organics who were given a series of trials with the spiral reported fewer after-effects for later than for earlier trials.

Eysenck and Eysenck (1960) made more specific predictions in relation to the general prediction made here. They predicted that a shorter rest pause (30 sec) should produce less reminiscence than a longer one (3 min) and that a larger number of pre-rest trials (12) should lead to a greater reminiscence than a smaller number (5). One minute exposures were given to 62 industrial apprentices. In this study, massed practice was given i.e. after the period during which the length of the after-effect due to previous stimulation was established, the next trial followed immediately. After the rest pause (following five or twelve pre-rest trials) six more massed trials were given. Reminiscence was scored by subtracting the last pre-rest trial from the first post-rest trial in the case of the two groups having five

pre-rest trials; for the other two groups the score was the mean of the last three pre-rest trials subtracted from the first post-rest trial.

It was found that in the case of the group with the 3 min rest period and the group with the 30 sec rest period, there was a highly significant decline in after-effect scores from trial 1 to trial 5; thereafter no further systematic change was detected. After the rest pause there is an increase in score for the 3 min rest groups and a further decline score for the 30 sec rest groups. Neither of the effects quite reached an appropriate level of significance on the *t*– test but the authors comment that the similarity of the graphs of the plotted results for both rest groups suggested that it was not a chance phenomenon. The differences in reminiscence produced by the two practice periods was not significant.

The authors also predicted that extraverts would show greater reminiscence effects than introverts. They obtained a significant correlation between the M.P.I. E score and reminiscence for the 12 trial 30 sec group ($r = 0{\cdot}55$) in line with prediction, but none of the other correlations were significant. It was also predicted that extraverts would show a greater decline in duration with massed practice. The correlation between E scores and a decline score (first pre-rest trial minus fifth pre-rest trial) was "positive as predicted but quite insignificant" ($r = 0{\cdot}14$). It is interesting in this connection also to note that the curves presented by Spivack and Levine (1959) showing the decline in duration of the after-effect with repeated stimulation suggests a greater decline for the brain damaged group than for the normal group. This is in line with the prediction from Eysenck's theory, but in view of the overall longer duration of the brain damaged group, must only be related to the prediction with caution.

The evidence then, with regard to the decline in duration as a result of successive stimulation is in line with the prediction, though Spivack and Levine's finding that the decline remained over a one month period suggests the possibility that other factors besides inhibition as used by Eysenck play a role. It has also been found that under certain conditions the initial decline may be followed by an increase in duration of the after-effect (Costello, 1960c), and further evidence of this was found in the second experiment to be reported here. The data with regard to reminiscence and the relation of both decline and reminiscence to personality differences are conflicting.

(5) It is predicted that with an increase in the duration of the stimulus, there would be an increase in the duration of the after-effect due to the build up of *X* and that this would reach an asymptote marking the point where satiation prevented the further accumulation of *X*.

Only Ehrenstein (1925) among the early workers appears to have mentioned that duration of the after-effect depends on stimulation time. Holland (1958) gave his subjects 15, 30, 60 and 90 sec stimulation and obtained the following mean duration times for the after-effect: 13·93 sec; 20·46 sec 22·21 sec; 25·17 sec. Although the data he reports do not indicate an asymptote in the duration of the after-effect, he reports in a footnote that further work indicated that the majority of subjects have reached asymptotic scores between 80 sec and 100 sec of stimulation. But Pickersgill and Jeeves (1958) reported an increase in duration of the after-effect over stimulation

times ranging from 5 sec to 10 min. They also report that subjects with persistent after-effects tend to show greater proportion as well as actual gain, e.g. between $2^1/_2$ min and 5 min, and 5 min and 10 min stimulation; there is a gain of more than $^1/_2$ compared with $^1/_3$ and $^1/_5$ respectively in those with very short after-effects. Spivack and Levine (1959) plotted the Median log duration of the after-effect against the exposure time (the exposure times used were 5, 10, 20, 30 and 50 sec) for a group of brain damaged and a group of normal subjects; they found that the duration of the after-effect increased with increase of stimulation and that the durations for the normals rose more steeply. Both groups from the graphs appear to reach an asymptote at 30 sec.

With regard to increase in duration with increase in stimulation, the data is in line with prediction, but there appear to be important individual differences. The data with regard to asymptotic values is conflicting.

(6) It is predicted that binocular stimulation by increasing X would produce longer after-effect durations than monocular stimulation.

The only data with regard to this prediction, in the writer's knowledge, is Pickersgill and Jeeves' (1959) who report that the duration of the after-effect for each separately is shorter than for both eyes together ($p < 0{\cdot}1$) which is in the right direction, but not statistically significant.

In summary then, it can be said that our first prediction, that extraverts would have shorter duration than introverts, is well supported. The evidence is in line with the prediction that depressant drugs would produce shorter after-effects than stimulant drugs. The evidence with regard to the effects of brain damage is conflicting, or, taking only the objective data, is in contraction to the prediction. As predicted, successive stimulation results in a progressive shortening of the after-effect though this may be followed by a progressive increase. There is no clear cut evidence with regard to reminiscence. The evidence is in line with the prediction that an increase in stimulation times results in an increase in duration of the after-effect, though data regarding asymptotes is conflicting. The prediction that binocular stimulation would result in longer after-effects than monocular is slightly supported.

Let us look for a moment at some other theories that have been put forward to account for the spiral after effect.

Wohlgemuth (1911) has given a detailed account of the early theories which he divided into three categories; (1) physical; (2) psychical; or (3) physiological. A brief account will be given here. With regard to (1), Purkinje (1825) used eye movements to explain the after-effect of watching cavalry go by, as did Adams (1834) to explain the after-effect of looking at a waterfall in Scotland. Lotze (1852) used unconscious eye movements to explain the effect when no fixation was used. Where fixation was used he had recourse to psychical factors. Helmholtz (1867) also had recourse to eye movements. Though the eye movement theory was not obviously wrong in connection with waterfall illusions (where movement was in one direction), it was difficult to use eye movements in explaining the after-effect of Plateau's spiral (1850) where movement was in a number of directions. Dvorak (1870) using superimposed spirals moving in opposing directions obtained

after-effects appropriate to the stimulation, which he rightly considered evidence against the eye movement theory. Kleinert (1878) and Wohlegemuth (1911) provided similar evidence against the eye movement theory.

With regard to (2), Zollner (1860) discussed the after-effect in terms of psychical factors. Wundt (1874) stating that the phenomenon was neither due to the effects of eye movements nor to "a mysterious reaction of the retina", put forward an explanation in terms of after-image aided by associative factors. Budde (1884) asks us to consider a point on the moving stimulus. Our attention, he says, jumps from the fixation point to the moving point. When the objective movement stops, our attention jumps ahead and the point appears to move backward. Wohlgemuth (1911) states that his findings that the after-effect is just as long under conditions where the subject's attention is engaged with mental arithmetic etc. argue against Budde's theory. We may say that the major criticism of such a theory is the difficulty of testing it. More telling with regard to psychical theories is Wohlgemuth's finding that a long contraction stimulation followed by a short expansion stimulation is followed by an expansion after-effect indicating a continuation of physiological effects from the first stimulation.

With regard to (3), Muller (1840) argued that since the after-images of the stimulus present an appearance of movement in the same direction as the stimulus, the stationary objects of the projection field will appear to move in the opposite direction. Ruete (1848) agrees with Muller. Oppell (1856) also agreed with Muller originally, though later rejected this theory in favour of a psychical type theory. Stern (1894) also favoured the Muller type of theory and went on to explain that after-effects perceived when the eyes were closed were due to the fact that entopic visions took the place of exterior objects.

Slightly different types of retinal theories were put forward by others. Mach (1875) writes, "Accordingly we have to think that with the movement of a retinal image, a special process is set up which is not present in the resting stage, and that in opposing movements similar processes in similar organs are excited but that the processes exclude each other in such a way that with the occurrence of one the other is counterbalanced and with the exhaustion of one the other occurs." Mach also states that this is not a theory but a "physical expression of observed psychical facts." Thompson (1877) talks in terms of retinal fatigue and the association of contrasts. Zehfuss (1880), impressed by the fact that the after-effect of looking out of a railway carriage window remained within the after-image of the window, concluded that the after-effect was a retinal one and talked in terms of the flow of blood across the retina. Tschermak (1881) discussed the after-effect in terms of the irregularity in the excitation at the anterior and posterior borders of the image which has moved across the retina. Heusse (1888) talked about nerve currents opposite in direction being caused by the stimulus in the retina. Hoppe (1894) proposed that on cessation of the primary movement, after-images are successively produced of the movement phases just passed, those of the last phase first, their summation causing the idea of a new movement.

Of all the above retinal theories it might be said that it would be difficult to test them and the evidence for central components outlined above suggests that none of them tell the whole story.

Other early physiological theories are: Classen's (1863) who looked for an explanation of the phenomenon in the reflex tendency of the eyes to follow any movement and the innervation of the antagonistic eye muscles to resist it. When the eye is turned to a stationary object, the increased innervation continues but being no longer adequate to the visual experience produces visual vertigo. Exner (1888) outlined an elaborate theory concerned with the sequence of stimulation in various nervous centres, and Wohlgemuth (1911) presents a similar theory.

Leiri (1928) presented a muscular type theory. He argues that the apparent approach of the rotating spiral produced an enlarged retinal image which becomes a stimulus for a reflex increase in accommodation. When the stimulation stops the reflex activity stops. There continues, however, an "accommodation innervation" which has been compensating for the reflex activity during rotation and which now produces the after-effect until the eye becomes adjusted to the new degree of accommodation. Gates (1934) rightly presents the opposing after-effects of two spirals moving in opposite direction as evidence against Leiri's theory, but her attempt to explain the effect in terms of changes in the "internal muscular systems of the mechanisms of accommodation and convergence" does not appear to be an adequate explanation.

More recently George (1953) has attempted to apply Osgood's and Heyer's (1952) statistical theory of figural after-effects to the spiral after-effect. Apart from Spitz's (1958) criticism that George's theory necessitates a test or projection surface with contours whereas the after-effect can be seen on a test surface devoid of contours, the theory could also be criticized on the grounds that assumptions are introduced with regard to the latency of fatigue effects which are not in the original theory.

Deutsch (1956) presents a wave theory. An impulse is said to be propagated from each part of a contour at right angles to its tangent. It is assumed that there is a refractory state in the conductivity of the tissue where a contour has been generating impulses. When a contour is in motion, the propagation of a wave front in the direction of the motion of the contour is accelerated. Concomitantly, the wave front propagated in the opposite direction would tend to be slowed down as it has to pass over an area through which a contour has most recently passed thereby causing a difference in the rate of travel of the wave fronts propagated backwards and forwards. When movement stops, this difference in rate of travel reverses itself since the forced speeding up in a particular direction leads to a refractoriness in that same direction. Furthermore, the wave front propagated in the reverse direction will no longer be passing over a space which has just been occupied by a contour. Spitz (1958) criticized Deutsch's theory also on the ground that contour lines are needed in the test or projection field. This does not seem to me to be so obviously necessary in Deutsch's theory. It is interesting that Deutsch (1959) reports a study in which a stationary spiral was viewed in a flickering light and appeared to be in motion. The apparent rotation

gave rise to an after-effect of an actually rotating spiral. Deutsch points out that the after-effect appears when the pattern remains stationary but the ground appears to spin, while Kohler's after-effects depend wholly on contours. In any case, with or without contours, from the statement that when a contour is in motion the propagation of a wave front in the direction of motion of the contour is accelerated, one would except a wave front to continue in the same direction in the beginning at least when the stimulation stopped. One can accept the proposition that wave fronts in one direction slow down wave fronts in the opposite direction but not so readily the proposition that forced speeding up in a particular direction leads to a refractoriness in the same direction.

Spitz (1958) attempts to provide a workable explanation of movement after-effects from the Kohler–Wallach theory of satiation without the addition of new concepts. He writes, "imagine that one is fixating upon a steady series of lines going from left to right. No matter where the lines are when they are first perceived, in those adjacent nerve fibres which occupy the area between the lines, the fibres on the left are always stimulated first. The 'left-sided' fibres are always one step ahead of adjacent 'right-sided' fibres. It is known from studies of stationary visual figural after-effects that after removal of the Inspection Figure, there is a recovery period during which the altered brain medium presumably regains permeability. In the present example, during the recovery period the left sided fibres will still be one stage ahead of their adjacent right-sided neighbours. This means that until there is complete recovery, the right-sided members will always be in a state of greater impedance and therefore perceived stimuli will flow to the left away from these areas of impedance." This looks all right, but we can just as well argue that since the right sided fibres are stimulated last, there will be residual excitation in that fibre when the movement is stopped and that movement will continue in the same direction from the left hand fibre that is satiated and in which the residual excitation has gone to the right hand fibre. Spitz himself presents a number of findings which cannot be accounted for by his theory and states that his proposal is presented "only as a preliminary hypothesis to be tested further and eventually modified." One wonders, however, what kind of prediction Spitz would make from his theory.

It may be of value to mention the study by Granit (1928). His hypothesis was that the greater, within limits, the retinal area affected by the primary movement produced by the revolving striped drum, the longer the duration of the after-effect. He found, however, that with an increase in the visual angle subtended beyond 4°, there was a decrease in the duration. He comments, "It seemed a highly probable assumption that the after-effect of movement in the rods exerts a restraining influence upon the development of the illusion of movement in the cones." In support of this hypothesis, he also presents evidence that impairment of the after-effect of seen movement at slight degrees of dark adaptation is relatively stronger than at greater degrees of dark adaptation.

But Grindley (1930) argued that on Granit's theory the ratio of the duration of the after-effect with the larger area to the duration with the

small area should be greater for red light than for light of other colours since when the larger area is stimulated by lights of other colours, there is mutual inhibition of the rods and cones, i.e. if the durations of the after-effect for the red light and the large and small retinal areas be called LR and SR respectively and those for the light of other colours LC and SC respectively, then on Granit's theory LR/SR/LC/SC $>$ 1. Comparing red and green lights the ratio obtained was 1·04, comparing red and violet lights it was 1·06, neither ratio being significantly different from 1. Grindley concludes that there is no satisfactory evidence of the interaction between rod and cone mechanisms in the production of the after-effect. He suggests that the explanation for Granit's results may be found partly in the changes in the perceptual configuration when the distance of the moving object from the observer is changed. Granit's finding that with an increase of distance of the stimulus, the after-effect was longer, he would interpret in terms of the smaller visual angle subtended. We have reported elsewhere the finding (Costello, 1960d) that a spiral at a greater distance resulted in a longer duration of after-effect than a photograph of the spiral nearer to the subject *but subtending the same visual angle.* This finding is more in keeping with Grindley's type of explanation than Granit's.

Now we will proceed to an account of the drug experiments using Eysenck's drug postulate as our guide.

The drug postulate is: Stimulant drugs increase excitatory potential and decrease inhibitory potential, while depressant drugs decrease excitatory potential and increase inhibitory potential.

The prediction is: The duration of the spiral after-effect will be decreased by the administration of meprobamate — a depressant drug.

The First Experiment

The first of two small preliminary drug experiments indicated that meprobamate, as predicted, decreased the duration of the after-effect. Meprobamate did not produce a significant reduction in the after-effect in the second preliminary study and this appeared to be due to the large individual differences in the reaction to the drug. These studies have been reported in detail elsewhere (Costello, 1960c).

The spiral used in all the studies is a four throw spiral of 180° (cf. Eysenck, 1953). It is $8^1/_4$ in. in diameter and is rotated by a variable speed electric motor controlled through a rheostat. The speed of rotation is set by means of a multiple speed strobe disk built into the back of the housing. The speed used in all studies is 100 rev/min. The distance of the spiral from the subject was 6 ft. Illumination for the spiral was provided by a 60 watt and 40 watt bulb mounted on a laboratory stand.

It was found in the preliminary experiments that a photograph of the spiral placed close to the subject tended to produce more reliable results when used as a projection field for the after-effect than when the spiral itself was used as the projection field.

The projection field in this study was a photograph of the spiral mounted on stiff black cardboard which was attached to the wall at the right

of the subject. The photograph was 3 in. in diameter and the distance from the subject was 27 in. Thus the photograph subtended a visual angle equal to that subtended by the spiral which was 6 ft from the subject. An adjustable chin rest ensured that the same distance was maintained from both the spiral and the photograph throughout the studies. Illumination for the photograph was provided by a 40 watt lamp 1 ft away.

After preliminary trials to familiarize the subject with the after-effect, the following instructions were given: "In a few seconds, I shall ask you to close your eyes and I shall set the spiral rotating. When I say 'Ready!', I want you to rest your chin on the chin rest, facing the spiral but with your eyes still closed. When I say 'Now!', open your eyes and fixate the screw in the centre of the spiral. After one minute, the spiral will stop rotating.

When the spiral stops rotating, the lights in front of the spiral will go out and the light on your right will come on. At that point, I want you to turn, keeping your chin on the chin rest, and look at the photograph of the spiral. You will experience an after-sensation of movement. It will be an expansion effect coupled with rotation in the opposite direction. When all the movement has stopped say 'Now!'."

The length of the after-effect was in all cases timed by a stop watch. Two trials were given. The stimulation period was 1 min and there was 1 min rest pause between trials.

Details with regard to subjects and times of administration of drugs, and times of testing will be found in the chapter on the after-image.

Each subject was given three treatments: (1) placebo; (2) 600 mg meprobamate and (3) 1200 mg meprobamate on three different days, the order of treatments being counterbalanced for subjects and for sex.

Results

Table 1 shows the result of an analysis of variance carried out to test the significance of the differences between after-effect scores for the three treatments.

It will be seen from Table 1 that five significant F ratios emerge, namely, those for "Subjects", "Treatment", "Subject/Treatment" interaction (residual 1), "Time" and "Subject/Treatment/Time" interaction. Fig. 1 shows the results in diagrammatic form. Neither the 'day' effect nor 'sex' effect contributed significantly to the variance.

A breakdown of the main treatment effect produces the following result:

	Mean after-effect
Placebo	18·29 sec
600 mg meprobamate	16·26 sec
1200 mg meprobamate	16·59 sec

The differences between placebo and 600 mg, and placebo and 1200 mg are significant at the 0·01 level, and the results are in line with the prediction that meprobamate would decrease the duration of the after-effect. The difference between 600 mg and 1200 mg is not statistically significant.

TABLE 1

Analysis of Variance of After-Effect Scores

Source	*Degree of Freedom*	*S. Squares*	*Mean Squares*	*F*	*P*
Subjects	17	11860·45	697·66	23·25	<0·001
Days	2	145·92	72·96	2·43	N.S.
Treatments	2	546·03	273·01	9·10	<0·001
Residual (1)	32	960·43	30·01	7·27	<0·001
Total	53	13512·63			
Time	7	332·77	47·54	8·38	<0·001
Subject/Time	119	818·21	6·88	1·21	N.S.
Day/Time	14	54·77	3·91	–	N.S.
Treatment/Time	14	97·67	6·98	1·23	N.S.
Residual (2)	224	1269·30	5·67	1·373	<0·01
Total	431	16085·35			
Final Residual (3)	432	1783·10	4·13		
Total	863	17868·45			
Between Sex	1	1820·63	1820·63	2·90	N.S.
Between Subjects within Sex	16	10039·62	527·48		

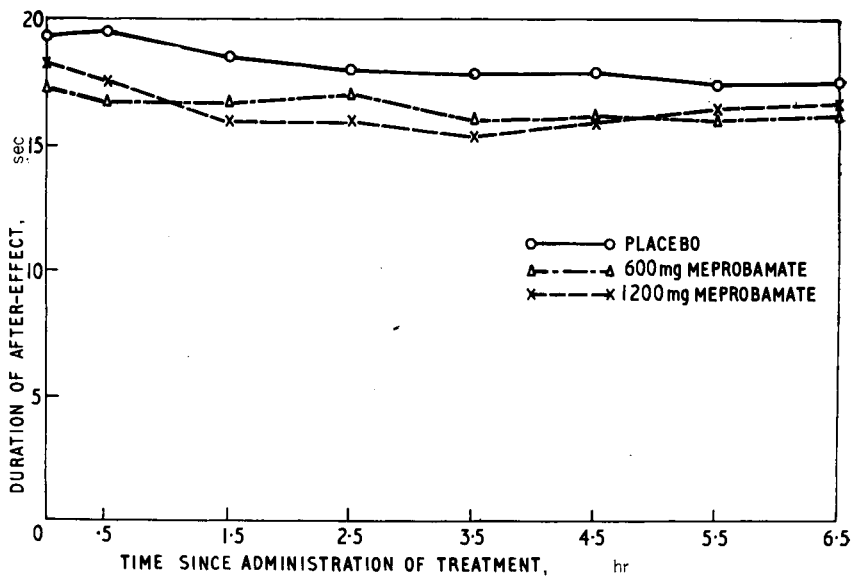


FIG. 1. Duration of the spiral after-effect for the group of subjects under drug treatment

The significant "subject/treatment" interaction indicates that the degree of meprobamate differs from subject to subject. As with the after-image, it was decided to correlate each subject's extraversion score with his percentage drop in duration of the after-effect after administration of the drug. The four scores of change are described in detail in the Chapter on the after-image and the results will be presented immediately here:

	Extraversion	
Spiral AE % decrease 600 mg (Max) − P(Max)	−0·43	
Spiral AE % decrease 600 mg (1 − 3) − P(1 − 3)	−0·31	
Spiral AE % decrease 1200 mg (Max) − P(Max)	−0·55	<0·05
Spiral AE % decrease 1200 mg (1 − 3) − P(1 − 3)	−0·60	<0·01

All the correlations are negative and two reach significance. Eysenck's theory predicts a negative correlation between extraversion and duration of after-effect. The E score for the subjects in this study was correlated with the mean of the two scores for the initial session of each subject's first day of testing, $r = -0{\cdot}11$ which is not significant. No predictions have been made by Eysenck with regard to the relationship between an individual's degree of extraversion and the degree of the drug effect, but as with the after-image we can say that if satiation builds up more readily in the extravert and our measures of change are measures of the satiating effect of meprobamate, then one would except a positive correlation between E and the measures of change. The results are contrary to the prediction.

The possibility that there was a relationship between the amount of decrease with the drug and the initial score was tested with the following result:

	Initial Score
Spiral AE % decrease 600 mg (Max) − P(Max)	0·06
Spiral AE % decrease 600 mg (1 − 3) − P(1 − 3)	0·20
Spiral AE % decrease 1200 mg (Max) − P(Max)	−0·23
Spiral AE % decrease 1200 mg (1 − 3) − P(1 − 3)	0·06

All the correlations are insignificant. The results suggest that the satiation produced by a depressant drug is a different process to that produced by stimulation with the spiral and extraversion appears to be more related to the former. Again, as with the after-image, we get a drop though not so marked in the test-retest reliability based on the initial scores ($r = 0{\cdot}95$) to that based on the scores $3^1/_2$ hours after administration of placebo ($r = 0{\cdot}84$), $3^1/_2$ hours after administration of 600 mg meprobamate ($r = 0{\cdot}86$) and $3^1/_2$ hours after administration of 1200 mg meprobamate ($r = 0{\cdot}80$).

The absence of a significant "treatment/time" effect is puzzling expecially in view of the results with the after-image and apparent movement (see the relevant chapters). It reminds us of the Spivack and Levine (1959) finding of a drop on the after-effect from one session to a second session 3 months later. At present, no explanation of the result can be given. It may be however that the significant "subject/treatment/time" interaction

is partly responsible. In any case, it was decided to investigate this second order interaction.

One would predict that the effect of the drug would occur earlier in extraverts since according to the theory, satiation builds up more quickly in extraverts. In other words, one would predict a negative correlation between E and the time to maximum effect, for 600 mg meprobamate r = 0·09, for 1200 mg meprobamate r = 0·00. Looking to weight as a possible important variable, it was found that for 600 mg meprobamate the correlation between time to maximum effect and weight was 0·20 for 1200 mg r — 0·037 both insignificant.

The correlation of the four measures of change with weight produced the following results:

	Weight
Spiral AE % decrease 600 mg (Max) − P(Max)	0·05
Spiral AE % decrease 600 mg (1 − 3) − P(1 − 3)	0·21
Spiral AE % decrease 1200 mg (Max) − P(Max)	0·01
Spiral AE % decrease 1200 mg (1 − 3) − P(1 − 3)	0·001

With respect then to the effects of meprobamate on the spiral after-effect we can tentatively conclude; (1) that meprobamate as predicted decreases the duration of the spiral after-effect, but that 1200 mg does not produce a significantly greater effect than 600 mg; (2) that extraversion correlates negatively with the amount of change produced by the drug; (3) that weight is not significantly correlated with the amount of change produced by the drug; (4) that the subject's initial score is not significantly correlated with the amount of change produced by the drug; (5) that the effect of the drug remains constant over the time used in this study; and (6) that neither extraversion nor weight were significantly correlated with the time to maximum effect of the drugs.

Correlations of the spiral scores with the after-image score and apparent movement scores were all insignificant. A discussion of this lack of correlation between the tests will be found in the chapter on the after-image.

A Comparison of the Effect of the Drug and of Massed Practice

Negative correlations were obtained between extraversion and the effect of meprobamate on the after-effect. If satiation produced by a depressant drug is similar to that produced by massed trials, one would predict a negative correlation between extraversion and the drop in duration of the after-effect due to massed trials.

Eysenck's theory as it stands at present would predict a positive correlation between extraversion and the drop in the duration of the spiral after-effect produced by massed trials. As we saw in the review, the evidence to date is conflicting.

We might ask also: Would the drop in duration of the after-effect due to massed practice be greater after administration of a depressant drug than after administration of a placebo or vice versa? Eysenck's theory would predict a greater drop after the depressant drug. But the prediction based

on the negative correlations between extraversion and the effects of meprobamate found in the previous study would be that there would be a greater drop after administration of a placebo.

The study to be reported was designed to test some of these predictions. The predictions will be listed in detail when we come to a discussion of the results.

Details with regard to subjects will be found in the chapter on the after-image. The stimulus conditions were the same as in the first study.

A detailed account of the experimental procedure will be found in the after-image chapter. It is sufficient to say here that each of the 20 subjects was given six massed trials followed by a 15 min rest pause followed by four more massed trials. This series of trials was given once on each of 2 days to every subjects. On one day the subject had a placebo, on the other day the subject had 800 mg meprobamate.

Fig. 2 shows the results in diagrammatic form.

(1) It is predicted that the duration of the spiral after-effect will be decreased by meprobamate. It will be seen from Fig. 2 that the results are in line with the prediction. The mean duration for the 20 subjects on the first post treatment trial is: on the placebo day, 21·47 sec; on the meprobamate day 19·36 sec. The difference between the means is significant at the 0·025 level on a one-tail test of significance.

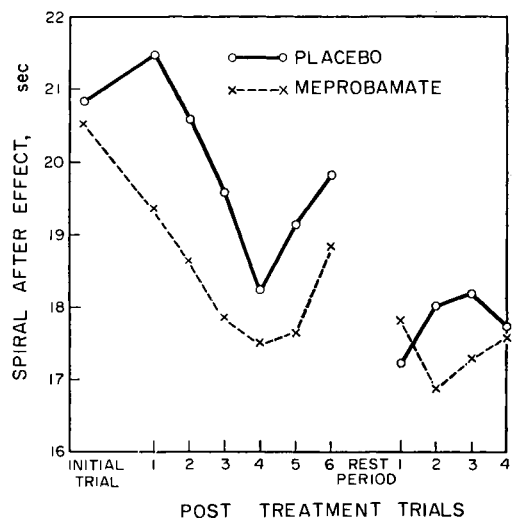


FIG. 2. The duration of the spiral after-effect for the group of subjects under drug treatment and conditions of massed trials.

(2) It is predicted, on the basis of the results in the first study, that there will be a negative correlation between extraversion and the percentage drop in the after-effect due to meprobamate. (It should be noted that, as in the first study, the percentage change produced by administration of the placebo

was subtracted from that produced by meprobamate in order to give a true measure of the drug effect.) The correlation obtained is 0·20 which is in the wrong direction but insignificant. Despite the lack of significance the result is surprising since the negative correlations between extraversion and the drug effect were obtained in the first study 1 $^1/_2$ hours after both 600 mg and 1200 mg meprobamate. In the first study, there was also a testing session ½ hour after the administration of treatment and it is possible that this is the crucial difference between the two experiments.

(3) It is predicted, on the basis of Eysenck's theory, that massed trials will produce a decrease in the duration of the after-effect. Looking at Fig. 2, we see that the results are by no means clearly in line with or contrary to the prediction. There is a decrease in duration on both placebo day and drug day from trial 1 to trial 4 (pre-rest), but, on both days, the duration begins to increase once more up to trial 6 (pre-rest).

Taking the results on the placebo day, the mean duration at trial 1 is 21·47 sec, at trial 4, 18·26 sec, the difference between the means being significant at the 0·01 level on a one tail test of significance. The drop on the drug day from 19·36 sec at trial 1 to 17·52 sec at trial 4 is significant at the 0·025 level (one tail). Eysenck's theory accounts then for the results under the conditions of this experiment for the first four trials.

The increase from 18·26 sec, at trial 4 to 19·85 sec at trial 6 on the placebo day is significant at the 0·025 level (one tail). The increase from 17·52 sec (trial 4) to 18·82 sec (trial 6) on the drug day is significant at the 0·05 level (one tail). These results cannot be predicted from Eysenck's theory at present.

If the increase in the duration of the after-effect from trial 4 to trial 6 on both days is due to processes similar to those that result in a decrease in duration from trial 1 to trial 4 on both days, one might expect a correlation between the drop from 1 to 4 and the increase from 4 to 6, though of course this is not absolutely necessary. The correlation between the drop from 1 to 4 and the increase from 4 to 6 on the placebo day is 0·15 which is insignificant. The correlation on the drug day is 0·505 which is significant at the 0·05 level. The results suggest that the processes responsible for the drop and the increase are similar. They also suggest the possibility of a homeostatic mechanism whereby, with repeated stimulation, satiation does not merely reach an asymptote but excitation sets in again. This increase in the duration of the after-effect after an initial decrease has been found in a previous study and the theory of homeostatic excitation has been outlined to account for it (Costello, 1960e). Some further remarks on the theory will be presented later.

These findings may explain the conflicting evidence in the work of Holland (1957), Pickersgill and Jeeves (1958), and Spivack and Levine (1959) with regard to the effects of increasing stimulation time, particularly in relation to asymptotes.

(4) It is predicted, on the basis of the findings of the main study, that there will be a negative correlation between extraversion and the decrease in duration due to massed trials. The correlation between E and the per cent drop from trial 1 to trial 4 on the placebo day is 0·00. On the drug day it is −0·46

($p < 0{\cdot}05$). That the result is in line with the prediction on the drug day but not on the placebo day, once again suggests that threshold values and possibly homeostatic mechanisms are of importance here.

Correlations were also calculated between E and the per cent increase from trial 4 to trial 6 on both days. On the placebo day, $r = -0{\cdot}13$. On the drug day, $r = -0{\cdot}08$. Both correlations, being insignificant, are suggestive of nothing.

(5) It is predicted, on the basis of Eysenck's theory, that the drop from trial 1 to trial 4 will be greater on the drug day than on the placebo day. The drop on the placebo day is 11·65 per cent, on the drug day 9·45 per cent. The difference between these mean percentages is insignificant. The tendency is contrary to that predicted by Eysenck's theory but in line with what would be predicted on the basis of the drug findings in the main study. The latter prediction assumes, of course, that satiation produced by a depressant drug is similar to that produced by massed trials. The correlation between the decrease in the spiral after-effect produced by the drug and the decrease from trial 1 to trial 4 on the placebo day is 0·29, which, though insignificant, gives, in its sign at least, some support to the assumption.

(6) It is predicted, on the basis of Eysenck's theory, that, after the 15 min rest interval, there will be an increase in the duration of the after-effect due to the dissipation of satiation. A glance at Fig. 2 will show that the results are contrary to the prediction. The decrease from trial 6 (pre-rest) 19·85 sec to trial 1 (post-rest) 17·26 sec is significant at the 0·01 level (two tails). The decrease from 18·82 sec to 17·82 sec on the drug day is not significant. The per cent drop from trial 6 (pre-rest) to trial 1 (post-rest) i.e. 12·3 per cent is significantly greater than on the drug day when it is 1·95 per cent.

The correlation between E and the per cent drop from trial 6 (pre-rest) to trial 1 (post-rest) on the placebo day is 0·14, on the drug day it is 0·48. ($p < 0{\cdot}05$).

One may ask whether or not the drop from trial 6 (pre-rest) to trial 1 (post-rest) is due to processes similar to those underlying the drop from trial 1 to trial 4 (pre-rest). The correlation on the placebo day is $-0{\cdot}14$ and on the drug day it is 0·19 which does not enable us to draw any definite conclusions.

The general finding of a decrease in the duration of the after-effect after the rest period is in keeping with Spivack and Levine's finding of a decrease in duration of the after-effect after a one month period (1959).

Some Comments on the Theory of Homeostatic Excitation

As we mentioned above, an account of the theory of homeostatic excitation has been presented elsewhere to account for the effects of massed practice and brain damage on the spiral after-effect and for the differences between the expansion and contraction after-effect (Costello, 1960e).

Shortly, the theory states that inhibition produced by stimulation builds up to a critical level beyond which further stimulation will result in an increase of excitation up to a second level beyond which inhibition will once more appear, and so on.

It is tentatively suggested that this reformulation of Eysenck's theory of excitation–inhibition may account for the negative correlation obtained in the first experiment between extraversion and the effects of meprobamate on the after-effect. What would be expected on the basis of the theory is a positive correlation between extraversion and the drug effect at low levels of drug (administration of low doses or testing a short time after high doses) and a negative correlation at high doses since the drug will result in the critical level of inhibition being reached in the extraverts. It will be remembered that though the negative correlations appeared between extraversion and all four measures of the drug effect ($1^1/_2$ hours after 600 mg and 1200 mg and maximum effect of 600 and 1200), the significant correlations only occurred with 1200 mg. In the second drug study, when 800 mg of meprobamate were used and there was no testing $^1/_2$ an hour after treatment, small positive correlations were obtained.

There can be no doubt from the above array of complex findings that future work with the spiral after-effect must be done over wider ranges of stimulation times and methods of administration in general.

The concept of homeostatic excitation, introduced in an attempt to explain some of the findings, may appear to have an *ad hoc* character, though it can be said that all scientific theories begin with the data they have to explain. But in order to take away some of this *ad hoc* character, let us look at the theory in a more general way.

One of the puzzling things about the effects of brain damage is the visual hallucinations that occur. Visual hallucinations, it is assumed, can be regarded as supervivid visual images. If the vividness of visual imagery is proportional to the excitatory potential, and there is some evidence for this (Costello, 1957), then why should hallucinations occur in those with brain damage, in whom, it is postulated, inhibitory potential is increased? Are the hallucinations the product of localized homeostatic excitation? The vivid imagery of hypnogogic phenomena that occur when falling asleep or when under the influence of depressants, may also be due to localized homeostatic excitation.

We refer to *homeostatic* excitation because of its apparent similarity to other homeostatic mechanisms. But unlike the homeostatic mechanisms that respond to slight variations of normal conditions, homeostatic excitation would appear to occur only under conditions producing excessive inhibition.

There is some similarity between the concept of homeostatic excitation and Pavlov's concept of protective inhibition produced by over stimulation (1927). It is a similarity of opposites, of course, since the writer's theory concerns excitation produced by over inhibition. Predictions from Pavlov's theory have been verified by Venables and Tizard (1956).

Pavlov also talks about "inhibitable type" dogs which after being made to stand still for long periods of time in experiments upon gastric secretion entered into "astonishing fits of excitation upon being released." "These wild attacks of excitation" wrote Pavlov "may possibly be regarded as a brief outburst of positive induction following a prolonged and intense inhibition." He mentions cases also in which conditioned reflexes were first of all inhibited by surgical lesions, but when they finally got re-estab-

lished, they were found not only to regain their normal strength but often to exceed it. Pavlov suggests the possibility that this is due to an increase in the intensity of the excitatory process.

Of interest also is Pavlov's discussion of positive induction. For instance, "a conditioned alimentary reflex is established to a rate of 76 beats per min of a metronome, and from this, a rate of 186 beats per min is completely differentiated both with regard to the secretory and motor components of the reflex. The positive stimulus tested immediately after the application of the inhibitory stimulus shows an increase of 30 per cent in the secretory reflex; the motor reaction was correspondingly intensified while the latent period of the secretory reflex was considerably shortened." A series of experiments on positive induction led Pavlov to the conclusion that, "induction makes its appearance only under the maximal development of a given cortical inhibition . . ." Sherrington's (1947) rebound exaltation following on inhibition is similar to Pavlov's positive induction.

None of the above types of excitation following on inhibition is precisely the same as the homeostatic excitation introduced here. But there are interesting similarities which give the theory perhaps more plausibility. But the *ad hoc* character of a theory can only be finally lost by the verification of its experimental implications and the theory presented has at least the merit of being able to produce testable predictions.

Summary and Conclusions

(1) A review of the literature on the spiral after-effect indicated that the spiral after-effect had important central components that the data available was in line with the predictions fom Eysenck's theory that:

(a) Extraverts have shorter after-effects than introverts. Significant correlations between extraversion and the after-effect have not been found isn this investigation due to the smallness and homeogeneity of the group used.

(b) That depressant drugs would reduce the duration of the after-effect. This has been confirmed in the two studies reported here.

(c) That successive stimulation would result in a decrease in the duration of the after-effect. This has been confirmed in part by the studies reported here. Reminiscence effects, predicted to occur after a rest period were not found. On the contrary, there was a further significant decrease in the duration of the after-effect after the rest period. The decrease in duration of the after-effect produced by massed trials may be followed by an increase in duration if stimulation is continued. Reference was made to a theory of homeostatic excitation which has been formulated in order to account for this finding (Costello, 1960e).

Negative correlations found between extraversion and the effects of massed trials were also explained in terms of the concept of homeostatic excitation.

(d) That with an increase in stimulation times there will be an increase in the duration of the after-effect.

(e) That binocular stimulation would produce longer after-effects than monocular stimulation.

(2) The review of the literature indicated that the data available was not clearly in line with the prediction from Eysenck's theory that the duration of the after-effect would be shorter in the brain damaged.

The fact that some investigators reported shorter after-effects and others longer has been explained elsewhere (Costello, 1960e) in terms of the kind of after-effect used (expansion or contraction), and the concept of homeostatic excitation.

(3) It was suggested that the negative correlations between extraversion and the drug effect found in the first experiment may be explained in terms of the concept of homeostatic excitation.

Chapter 9

THE EFFECTS OF STIMULANT AND DEPRESSANT DRUGS UPON VISUAL FIGURAL AFTER-EFFECTS

H. C. HOLLAND and BEATRIZ HERRERA GOMEZ*

The phenomenon of figural after-effect which consists of the distortion of sensory input, subsequent to and consequent upon preceding stimulation in the same area** has been known by this name since about 1944 when Kohler and Wallach (1944) collected most of the existing data on similar effects in perception (notably by Gibson, 1933; 1937) and published, under the title of "*Figural after-effects: an investigation of visual processes*," an experimental and theoretical account of the phenomenon in vision. A tremendous literature has already appeared and is still appearing, which in size is perhaps second only to that concerned with critical flicker fusion, and many parameters of the phenomenon have been outlined. Several similar phenomena which fulfil the essential stimulus requirements of the effect have been demonstrated in hearing (Deutsch, 1951; Jones, 1949, Krauskopf. 1954, in Kinesthesis, by Taffe, 1951, Klein & Kretch, 1952 Eysenck, 1955), and in autokinesis (Crutchfield, 1949), and their effect demonstrated in a number of other phenomena. Figural after-effects have been advanced, and largely accepted, as illustrating a general principle of the mechanisms of perception, namely, that the perceptual resolving structures are subject to displacement.

The underlying mechanism of figural after-effects is satiation, a process which results in a condition of the mediating neurone which is similar to a localized fatigue effect in the sensory projection areas. One of the basic assumption of the theory is that the stimulus figure is isomorphically projected on the cortex, producing, coincident with its contours, a state or condition of increased resistance to subsequent stimuli in the same area. The polarizability of the affected membrane is lowered and subsequent stimulus effects, figure currents, are displaced away from the satiated region into one of less resistance or higher response potential.

As we remarked earlier, the impressive literature dealing with figural after-effects refers to many investigations which have studied and attempted to delineate the specific parameters of the phenomenon, including size, brightness, dimensionality and a large number of different aspects have been studied. Reviews of these studies are readily available, (cf. McEwan, 1958; Day, 1959) and it would be repetitive to outline them here. Apart from this

* The writers are indebted to the Wallace Laboratories for support.

** Although "area" is, strictly speaking, a locational concept, this statement would hold whether psycho-architectural or psycho-spatial isomorphism is implied.

we are concerned primarily in this experiment with only two major contributing variables, namely, the temporal aspects of short duration stimulation and the effects due to individual differences, more specifically to drugs x individual differences.

Temporal Factors

One of the first and most important questions asked of figural after-effects was: "how does the magnitude of F.A.E. vary as a function of the preceding stimulation, i.e. the inspection figure?" Perhaps the first attempt to present some kind of answer to this question lies in an experiment by Hammer (1949), although Gibson (1937) had outlined a basically similar function for tilted lines. Hammer conducted three experiments measuring the growth and decay of figural after-effects for different fixation periods and different intervals of delay between the offset of the inspection figure and the onset of the test figure. It is an early work, based upon only three subjects but it is still referred to as the "most careful estimate of this function" (Day, 1959). Hammer's work was such as to indicate that a recognisable figural after-effect was observable after about 5 sec of inspection and that this increased up to about 1 min, the effects of stimulation lasting about $1^1/_2$ min. One of the weaknesses of Hammer's study lies in the psychophysical method employed, a method of limits. This has been corrected in more recent studies (cf. Krauskopf, 1954) particularly in Japan. The method has been changed to the somewhat longer but more accurate form, the "constant method".

A large number of studies on the psycho-physical aspects of figural after-effects has been conducted by Japanese authors and it is from these that much the best recent work emanates. A recent review by Sagara and Oyama, (1957), written specifically to assist English speaking perceptionists, summarizes the results of many experiments. Of particular interest here are the experimental findings of Ikeda and Obonai, (1953a; 1953b; 1955*;) whose elegant experiments are well known in this field. These authors presented inspection figures for inspection periods from 1 sec to 240 sec (i.e. 1, 5, 15, 30, 60, 120, 240 sec) and discovered that the magnitude of the figural after-effect remained pretty much the same for any length of inspection. The only difference between the longer and shorter periods was that the former produced more persistent after-effects.

As far as the present research is concerned, the most important aspect of Ikeda's (1953) study lies in the fact that inspection periods as short as 1 sec were long enough to produce a clearly observable figural after-effect. This period is probably as short as any in the current literature. Recently Duncan (1958) has employed a fixation period of 4 sec but failed to obtain any consistent effect. The present experiment attempts to elucidate further the temporal aspects of figural after-effects by covering the time periods outlined by Hammer's and by Ikeda's studies, and to examine inspection periods below 5 sec.

* The authors would like to take this opportunity to express their thanks to Hisako Ikeda, now a member of the staff at the Institute of Psychiatry, for many interesting discussions about F.A.Es.

Individual Differences

It has repeatedly been observed, even in the original studies of Kohler and Wallach (1944), that there are considerable individual differences in figural after-effects. Few studies, however, have deliberately investigated them and there are still fewer explanations of their causes forthcoming. The following studies are exceptions to this generalization. Klein and Kretch, (1952) using kinaesthetic figural after-effects have modified the original satiation theory to an assertion based on cortical conductivity and have demonstrated their prediction that there are differences between brain damaged and normal subjects. Almost simultaneously, however, a study by Jaffe, (1954), which had several weaknesses, failed to discriminate adequately between samples of the same two groups. One of the first to offer a coherent explanation of individual differences in figure after-effects was Eysenck, (1955) again employing the kinaesthetic phenomenon. Eysenck made the same prediction of hysterics as Klein and Kretch had of organics ... his theory of personality asserting, and his work indicating, several similarities between the performance of these types. Unlike Klein and Kretch, however, Eysenck did not accept the concept of cortical conductivity but asserted the essential similarity between "satiation" as defined by Kohler and "reactive inhibition" as defined by Hull (1943). This concept of reactive inhibition is, of course, one of the corner stones of Eysenck's theory of personality but, up to this rapprochement, had been mainly relevant to a response oriented theory of behaviour. Through the theory of satiation, it was clear that "inhibition" was applicable to perceptual phenomena. This relevance was clarified a few years later by Duncan's (1956) closely reasoned assertions which outlined the parameters of the similarities Eysenck had only briefly indicated.

Although intrinsically capable of very precise prediction and testing, the relationship between extraversion (the behavioural aspect of inhibition) and satiation (the perceptual aspect of inhibition) has not emerged with any great clarity from studies aimed at its demonstration. For instance, Nichols, (1956) administered a large battery of satiational indicants which failed to discriminate between extraverted and introverted neurotics, even though several of the individual tests, including kinaesthetic figural after-effects, did so. Holland (1959) also had only very limited success with his battery of tests used to test out an almost identical hypothesis to that of Nichols.

Several reasons for the limited succes of their studies were advanced by both Nichols and Holland. One of these was based on the duration of fixation required by tests of continuous stimulation. In consultation with Eysenck, who supervised both investigations, it was suggested that the several minutes fixation of inspection figures (if it is subject to inhibition) would in itself be different in the two personality types, and this would militate against differential figural after-effects. In Holland's (1959) study, a putative test of fixation was included but failed to correlate with the magnitude of after-effect. It was concluded that what was required was some method of inducing figural after-effects which would not capitalize

upon differences in the ability of subjects to maintain accurate and sustained fixation.

In the field of figural after-effects and drugs the literature is not extensive. Wertheimer (1954a; 1954b; 1955a; 1955b) in a series of investigations, has approached the study of individual differences in figural after-effect by the use of an electro-chemical concept which asserts that differences in effects represent differences in the modifiability of cortical conductivity which in turn is related to metabolic efficiency (Wertheimer 1955a). He has supported his explanatory theory both by the use of metabolic drugs and by the use of a criterion group of schizophrenics exemplifying small figural after-effects and low metabolic rate (Wertheimer, 1954a). Like Eysenck's, Wertheimer's theory would demand the inter-correlation of figural after-effects from different modalities and, indeed, he has produced this. Eysenck's students, on the other hand, have failed to do this convincingly as yet (cf. Nichols and Holland). Eysenck's approach to the effect of drugs differs from Wertheimer's. He employs an explanation of drug effects on behaviour of which the reader is, no doubt, aware and which is outlined in greater detail elsewhere in this volume. Briefly the drug postulate asserts that the depressant compounds will increase inhibition and decrease excitation whereas the stimulants will decrease excitation and increase inhibition, thereby increasing and decreasing extraversion respectively. In its application to figural after-effects the drug postulate is quite clear. Above we have seen that figural after-effects are the perceptual aspects of inhibition and we now see that depressant and stimulant drugs are a means of manipulating the inhibitory balance (inhibition – excitation). Clearly, therefore, stimulant and depressant drugs should decrease and increase the magnitude and persistence of figural after-effects. This prediction is one which has been tested in a number of experiments on perceptual phenomena where a satiation hypothesis is directly or indirectly applicable. Eysenck (1960) gives the sources of most of these, cf. also Eysenck (1957).

To summarize this general introduction so far, we may say as follows: there are two aims to the present study (a) an examination of short exposures on visual figural after-effects, and (b) a contribution to the validity of the extraversion theory through the drug postulate and its application to satiation. The combination of these two aims into the same experiment is particularly useful in the light of the reasons advanced (differential effects of fixation) for the limited success of previous studies to establish clearly the relationship between extraversion and visual figural after-effects, and the drug aspect offers us an alternative approach to the personality hypothesis.

Apparatus

The apparatus is essentially the same as that already described for the investigation of visual masking outlined in Chapter 3.

The only modification was the addition of timing capacitors to increase the exposure intervals from 300 μsec to 9 sec for the light stages and from 300 μsec to 6 sec for the dark and recycle stages. The difference, apart from this, lies in the presentation of the stimuli. The inspection figure was

a black outline circle on a white card, its inside diameter being 6 cm and its outside diameter 7 cm. The inspection figure was viewed through the half silvered mirror of the tachistoscope. The test figures numbered eight. They were mounted upon two 20 in. diameter duralumin "wheels" faced with white drawing board (Bristol Board). These wheels or disks could be rotated to bring into the same phenomenal disposition as the inspection figure, but reflected through the mirror, any one of the eight pairs of test stimuli. The test stimuli had one circle (the one which fell within the inspection circle) always the same diameter of 5 cm and this was paired with another circle which was one of the following sizes: —4·1, 4·2, 4·3, 4·4, 4·6, 4·8, 5·0 and 5·1 cm. Both test stimuli were black outline circles on white backgrounds v.a. = 4·00°. The thickness of the circles was 1 mm. By the method of constant stimuli, the subject was asked to report whether the "left" or "right" circle of the pair was the smaller. If the left test stimulus (5 cm) was judged smaller, a figural after-effect was existent and its size was the difference between it and its smaller companion. Thus, the maximum figural after-effect which could be determined by this method was 9 mm (5·0—4·1 mm.) and the maximum visual angle of the difference was approximately $^1/_2°$.

Procedure

Inspection figure — The inspection figure was exposed for six different time periods, i.e. 5, 3, 1, $^1/_2$, $^1/_4$, $^1/_6$ sec.

Interval — The interval between the offset of the inspection figure and the onset of the test figure was 100 msec.

Test figure — The test figures were exposed for a fixed time interval of 200 msec.

Control P.S.E. — Before commencing the test proper, a (control) poin- of subjective equality was assessed as the average of two series of presentations of the test figures without the inspection figure.

As remarked above, the problem for the subject lay in the simple judgement of which of the pair of test circles was the smaller. The six inspection figure intervals were randomized in accordance with a 6×6 Latin square design to compensate for differential practice effects. The specific order of presentation was chosen by each subject on the first day by selecting a number from a box. Once chosen the same order was retained on the 3 days.

A specific trial took the form of a joint stimulus presentation and judgement followed by an interval of several seconds. The interval between individual trials was assessed subjectively but was approximately 5 sec. As we have also remarked above, the eight pairs of test stimuli were mounted in alternative order upon 2 disks. The disks were also alternated. They were changed at the midpoint when half of each of the six sets of trials had been undergone. When the whole of one complete series of trials was finished (48 presentations) a 5 min rest interval was taken before the procedure was re-

peated. Each days total testing comprised three series of 48 presentations with rest intervals between. The thresholds counted as the results of the experiment were the average of the three series, each series being of eight trials.

Testing was conducted in a darkened room. The subject fixated a small red pin-point of light during testing.

Drugs

The three drug treatment were as follows:

Sodium amylobarbitone, 290 mg.
Dexamphetamine sulphate, 10 mg.
Placebo (starch B.P.).

The drugs were administered in identically appearing capsules, and taken orally. There were two capsules to each dose. The subject was tested not less than one hour and not more than 2 hours after ingestion. Drugs were administered in accordance with a 3×3 Latin square. With the exception of one subject, the double blind method was used for dispensing the drugs.

Subjects

There were 12 subjects in all. They were both male and female volunteers. Their ages ranged from 19 to 34 years. They were examined by a physician daily, before and after testing.

Results

The general outline of the results may be seen by reference to Table 1 and Fig. 1, which require little preparatory discussion or evalution. They

TABLE 1

Table showing the magnitude of displacement (F.A.E.) for the six times of inspection intervals for the three drug treatments. Below the displacement magnitudes are presented first the differences in size of F.A.E. from the depressant drug condition to the stimulant drug condition, followed by the percentage of this change as a function of the overall mean F.A.E. scores are in millimeters

	5 sec	*3 sec*	*1 sec*	*0·500 sec*	*0·250 sec*	*0·150 sec*	*Mean*
Amytal	3·04	2·54	1·68	1·93	1·34	1·33	1·977
Dexedrine	2·10	2·17	1·11	0·39	0·22	0·77	1·127
Placebo	1·02	1·33	0·72	1·10	0·15	0·87	0·865
mean	2·053	2·013	1·170	1·140	0·570	0·990	1·323
Visual angle	0·114°	0·112°	0·065°	0·063°	0·032°	0·055°	0·073°
Displacement	6·8′	6·7′	3·9′	3·8′	1·9′	3·3′	4·4′
Amytal - dex difference	0·94	0·37	0·57	1·54	1·12	0·56	0·850
Percentage of total	46	18	48	135	196	57	64

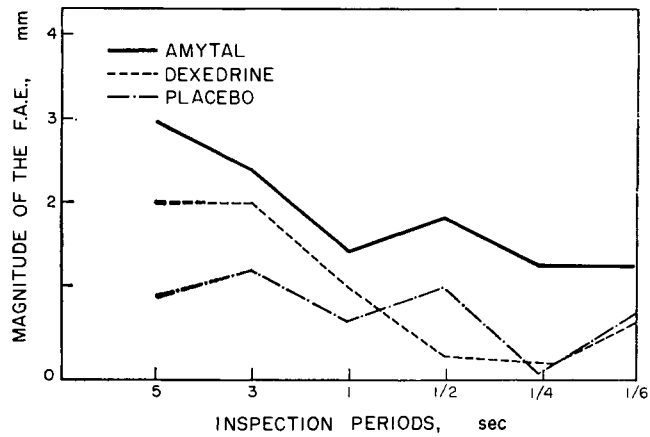


FIG. 1. Graph outlining the magnitude of visual figural after-effect in relation to the length of the inspection period.

outline the form of the declining function of the after-effect as it decreases from a little over 3 mm at the longest inspection period to a little less than 1 mm at the shorter intervals. The distances quoted represent approximate displacements of 10' and 3' respectively.

The differences between scores were assessed by analysis of variance in accordance with the logic of the experimental design. Its outline is presented in Table 2. Table 2 delineates three significant sources of variance as main effects. All three are very highly significant by variance ratio when tested against error. The first source is that presented as differences

TABLE 2

Outline of analysis of variance of figural after-effect scores for the six inspection periods and the three drug treatments

Source	*D.F.*	*S.S.*	*M.S.V.*	*F.*	*P.*
Subjects	11	180·69	16·43	15·35	1%
Insp. periods	5	62·58	12·52	11·70	1%
Drugs	2	48·54	24·27	22·68	1%
Orders	5	8·23	1·65	1·54	N.S.
Interaction People × Drug	22	175·60	7·98	7·46	1%
Interaction Insp. periods × Drugs	10	17·73	1·77	1·65	N.S.
Residual	161	171·64	1·07		
Total	216	665·01			

between "people" and represents individual differences, the second is due to the differences between "inspection periods" which are self explanatory, and the third is due to the drug effect. The interaction between people and drugs is also highly significant.

The overall differences between the main effects have been broken down into individual comparisons and these are presented in Table 3.

TABLE 3

Breakdown of overall variance into individual comparisons between "Inspection periods" and "Drugs"

$$t = \frac{\bar{x} - \bar{y}}{\sqrt{Se^2\left(\frac{1}{Nx} + \frac{1}{Ny}\right)}}$$

Inspection periods

$\bar{X}_5$	$\bar{X}_3$	$\bar{X}_1$	$\bar{X}_{500}$	$\bar{X}_{250}$	$\bar{X}_{150}$
2·05 mm	2·01 mm	1·17 mm	1·14 mm	0·57 mm	0·99 mm

$t_{5\times3}$, = 0·17; NS
$t_{5\times1}$, = 3·82; 1%
$t_{5\times500}$, = 3·99; 1%
$t_{5\times250}$, = 6·43; 1%
$t_{5\times150}$, = 4·65; 1%

$t_{3\times1}$, = 3·65; 1%
$t_{3\times500}$, = 3·78; 1%
$t_{3\times250}$, = 6·26; 1%
$t_{3\times150}$, = 4·43; 1%

$t_{1\times500}$, = 0·13; NS
$t_{1\times250}$, = 2·61; 1%
$t_{1\times150}$, = 0·78; NS

$t_{500\times250}$, = 2·47; 2%
$t_{500\times150}$, = 0·65, NS

$t_{250\times150}$, = 1·84; NS

Drugs

	Amytal (A)	Dexedrine (B)	Placebo (C)
$\bar{X}$	1·98	1·13	0·87

t_{AB}, = 2·66; 1%
t_{AC}, = 3·47; 1%

t_{BC}, = 0·81; NS

They show that the longer intervals (5 sec and 3 sec), with only one contradiction, are clearly and very highly differentiated from the remainder. Below the 3 sec period, differences are less clearly defined and there is a tendency for the function to increase at the shortest intervals.

The total drug variance is also broken down and presented in Table 3. Here individual comparisons between treatments show that Amytal is clearly and significantly differentiated from both Dexedrine and placebo but that Dexedrine in turn is not differentiated from placebo at an acceptable level.

DISCUSSION

It must be concluded that the experiment has been successful on most points, even though there are one or two particulars wanting. In terms of the temporal aspects, the results indicate that it is possible to obtain reliable figural after-effects with very short durations of exposure to the satiating effects of inspection figures. The results have also confirmed earlier work which demonstrated after-effect with inspection intervals of 5 sec (Hammer) and 1 sec (Ikeda) and have extended these demonstrations of the speed of polarizability to durations as short as 150 msec. The visual angle of the recorded displacements are also consistent with other work.* The shorter intervals mentioned are of particular interest in the light of the controversy which continues over the two major theories advanced to meet the phenomenon of perceptual displacement i.e. the "satiation" theory and the "statistical" theory.

As we have seen above, the satiation theory offers perhaps the best conceptual explanation of sensory displacement but is suspect on the grounds of its postulation of an unorthodox neurophysiology (a homogeneous cortical medium) and the lack of universality (cf. Marks, 1949). The statistical theory (Osgood, 1953), on the other hand, although orthodox, has an application and a relevance to other phenomena (eg. auditory F.A.E.) which are, to say the least, obscure. Short exposures may prove to be a method of investigating the difference between the two theories as in this respect, they should generate different predictions. The satiation theory has little to say about the temporal aspects of satiation except in terms of effects. Several studies, however, have emphasized the fact that satiation effects appear immediately after the removal of the inspection figure and persist in time as a function of the interval of stimulation. Statistical theory assumes that perceptual displacement is due overlapping distributions of excitation from the inspection figure and test figure, this in turn being due to the "blurring" of the sensory event by physiological nystagmus. The nystagmus is composed of movements which are part of a general distribution of 2′ of arc 10–100 per sec, 4′ of arc 5 per sec, and about once per second as much as $^1/_2$ a degree. It, therefore, seems feasible to present inspection figures at durations which would not permit of the establishment of any such excitatory distribution as is demanded by the statistical theory, and which would permit an *experimentum crucis* of the two alternative views.*

The main experimental hypothesis concerning drug effects is confirmed although the prediction was imperfect in every particular, e.g. the result of the placebo treatment. Implicit in the use of a placebo is the assertion that it controls for a "pill taking" effect and in some way represents the "normal" response. Unfortunately, this is not always the case and by administering the pill as a control against direct suggestibility, one can so easily

* Work by a number of authors shows that displacement magnitudes are between $1^1/_2$–8′ of visual angle.

* We have obtained measurable figural after-effects with inspection intervals as low as 5 msec and we hope to expand this work in the future.

fall foul of the effects of indirect or secondary suggestibility. It is, of course, possible to control drug experiments for both direct suggestibility and pill taking effects, but this increases the experimental population of a factor (3×3 becomes 4×4) and is usually non-economical. In this experiment the placebo effect is such that no explanation will be offered.

Apart from the placebo effect, the prediction based upon the drug postulate is supported at a very high level. The results must also be looked upon as lending support to those views expressing the relative importance of differential fixation as one of the parameters of differential figural after-effect. An examination of Table 1 also shows that there is a tendency for the drug effect to increase as the inspection period becomes shorter. The results by no means confirm the hypothesis, but the present support of the drug postulate when short exposures were used, is in contradiction to the failure which attended a similar drug study using several minutes fixation (Eysenck and Easterbrook 1960) and must be taken as supporting the view that extraverts retain fixation attention less well than introverts.

Summary

An investigation is reported which aims at extending our information concerning the duration of inspection required to produce demonstrable and reliable figural after-effects. A second aim was the confirmation or denial of a prediction based upon the effects of stimulant and depressant drugs derived from the extraversion theory. Incidental to these main studies was the examination of the hypothesis that shorter inspection intervals were more likely to be effective in the demonstration of differential satiation effects.

Results have confirmed the main extraversion drug prediction and also indicated that figural after-effects can be obtained at shorter intervals than employed before. This latter finding is thought to be relevant to the satiation–statistical theory controversy. The confirmation of the drug prediction at such a high level of confidence lends support to the asserted relationship between satiation and reactive inhibition.

Chapter 10

THE EFFECTS OF MEPROBAMATE ON APPARENT MOVEMENT

C. G. COSTELLO*

An account of the general framework of the author's drug experiments and a review of the literature on meprobamate will be found in the chapter on the after-image.

We shall go straight to a review of the relevant literature on apparent movement.

Review of the Literature

We are concerned with two questions (1) what is the evidence that the apparent movement phenomenon is a central rather than a peripheral phenomenon? (2) How far can the data on the test be predicted from the theory of excitation–inhibition?

A general discussion of the central–peripheral problem will be found in the chapter on the after-image. We shall use the same four criteria in deciding whether or not apparent movement is a central rather than a peripheral phenomenon. We shall, in other words, decide that central factors play an important part in determining the threshold of apparent movement:

(1) If we can show that stimulation presented to one eye has an effect on the threshold of apparent movement produced by stimulation to the other eye, or if apparent movement phenomena produced by binocular stimulation are different to those produced by monocular stimulation.

(2) If we can show that physiological stresses — drugs etc. — which have an effect on areas other than the retina produce a change in the threshold of apparent movement.

(3) If changes in the threshold of apparent movement can be shown to be related to measurable cortical processes.

(4) If differences in the threshold of apparent movement can be shown to be related to measurable differences in personality.

Interaction between Stimulation to Both Eyes

Shipley, Kenney and King (1945) reported that *phi* movement was perceived when stimulation was monocular, binocular and interocular and that binocular stimulation was the most favourable condition for the appear-

* The writer is indebted to the Wallace Laboratories for support.

ance of the phenomenon. K.R. Smith (1948) also reports that apparent movement was perceived with interocular presentation. Gengerelli (1948) presented four points in the form below:

$$\begin{matrix} p_2 & p_1' \\ p_1 & p_2' \end{matrix}$$

With visual fixation at the middle of the square constituted by the four points, the dimensions of the square were such that points p_1' and p_2' fell on one side of the retinal midline and points p_1 and p_2 fell on the other. In this way, stimulation from the primed points went to one cerebral hemisphere and that from the unprimed points went to the other. With presentation of p_1 and p_1' at t_1, and p_2 and p_2' at t_2, it was found that reciprocal vertical apparent movement predominated over reciprocal horizontal apparent movement from which Gengerelli concluded that the intercorrelation between the homonymous points is stronger than between the heteronymous points. When the visual fixation was to one side of the square of four points so that only homonymous cerebral excitations were present, there was no clear predominance of one type of movement over the other. Ammons and Weitz (1951) comment that "one important variable was not investigated, that is, the relative role of retinal factors in mediating the *phi* phenomenon." Though this is true, there is no reason to suggest that retinal factors played a role in producing the significant differences Gengerelli reports.

Ammons and Weitz (1951) designed an experiment which would present data with regard to the effects of exciting one or both cerebral hemispheres neglected by Shipley, Kenney and King (1945) and with regard to the importance of retinal interaction neglected by Gengerelli (1948). They found that apparent movement was reported in all conditions of observation and conclude that interaction possibly occurs at the cortical level, though they stressed the importance also of retinal or "at least subcortical factors." The latter point was stressed in view of the fact that the frequency of apparent movement perception is greater with monocular stimulation than with binocular and also in view of the fact that, with monocular stimulation, a higher frequency of apparent movement was reported where the stimuli were farther out in the periphery than when they were more central.

Deatherage and Bitterman (1952) also obtained apparent movement with interocular stimulation.

It may be concluded that the evidence satisfies the criterion.

Effects of Stresses Acting on Areas Other than the Retina

There is a major problem in the assessment of reported findings here. We must distinguish between the effects of stresses on psychological factors such as set, interpretation of meaning etc., which may be related to the perception of apparent movement and the effects of stresses on physiological processes underlying the apparent movement. It is obvious that only positive findings in the latter area can be taken as positive evidence

in relation to our criterion. Reference will be made to this problem again later.

Smith and Kappauf (1940) reported that cats, in which there was bilateral ablation of the visual cortex, when presented with a striped drum situation producing stroboscopic movement, showed similar nystagmic movement to humans in the same situation with slow component in the direction of the apparent movement reported by human subjects. Smith (1940) also found that bilateral decortication did not affect the nystagmic movements of the heads of guinea-pigs when presented with the striped drum situation. Though Smith concludes that cortical centres are unnecessary for apparent movement, we must regard such evidence as neutral with regard to our criterion since (1) it is dangerous to generalize from such studies to human subjects, (2) the presence of apparent movement in the absence of cortical centres does not rule out the possible influence of such centres when they are present.

Bender and Teuber (1949) report that apparent movement is not perceived in the affected fields in cases with localized cortical lesions.

Werner and Thuma (1942) reported findings for 20 brain damaged subjects and 20 endogenous mental defectives. When abstract figures (parallel lines and a right angle) were presented with time intervals ranging from 0 to 1175 msec, 18 of the 20 brain damaged subjects failed to report apparent movement whereas all the control subjects reported apparent movement. When a clock figure was used such that apparent movement would give the appearance of a pendulum swinging to and fro, the difference between the groups disappeared – both groups reporting apparent movement. The fact that Brenner has shown that, under Werner and Thuma's conditions, the movement is rationalized – it is inferred after the perceptual event has been completed suggests that we cannot accept this evidence as pro or con the criterion under consideration. Brenner (1953) also found that under conditions where the apparent movement is rationalized or inferred, brain damage has an effect but not under conditions of visible apparent movement. The latter finding we must consider as negative in relation to our criterion.

Saucer and Deabler (1956) reported findings with regard to the rate of flashes per sec of two lights at which *beta* movement disappeared. They tested 10 organics, 10 schizophrenics, 10 lobotomized subjects and 10 controls. The mean rates were, organics 3·58; schizophrenics 3·96; lobotomized patients 4·45; controls 4·52. The organics were significantly different from lobotomized patients and normals but not from schizophrenics. Since the authors were working with *beta* visible movement and not inferred movement, we can accept the data as being in agreement with our criterion.

Saucer and Coppinger (1961) found that whereas all their normal subjects ($N = 54$) perceived apparent movement, 30 per cent their non-psychotic hemiplegics ($N = 23$) failed to perceive apparent movement under any set of conditions.

Most of the data then, must be considered as neutral with regard to the criterion; Brenner's data is negative, Saucer and Deabler's (1956)

and Saucer and Coppinger's (1960) positive. The criterion is not definitely satisfied.

Relation of Apparent Movement to Cortical Processes

With regard to this criterion there is no evidence to the writer's knowledge. Indeed it would be difficult to relate it to the *alpha* rhythm of the EEG since the necessity of having the subject's eyes open would result in many cases in a blocking of the *alpha* rhythm. There is, however, Brenner's findings (1957) that apparent simultaneity and apparent movement are perceived at slower frequencies by younger children which confirms the results of Meili and Tobler (1931). Brenner comments, "In considering apparent movement perception only, it is interesting to note that both the mean flash frequency as well as its modification as a function of age is not unlike the frequency of the dominant brain rhythm found in the EEG's of normal children."

The Relation of Apparent Movement to Personality Differences

With regard to this criterion there is no evidence to the writer's knowledge.

Only in the case of the first criterion then is there a substantial amount of evidence. Since the first criterion has been satisfied, we shall tentatively accept that apparent movement has important central components.

We must now consider our second question: Can the data available be predicted from Eysenck's theory of excitation–inhibition? Since Eysenck himself has not given a sufficiently detailed theoretical formula from which predictions can be made, it will be necessary to do so here.

Although if one assumes that irradiation processes are involved in the perception of apparent movement one can make the general prediction from Eysenck's theory that apparent movement thresholds will be affected by the excitation–inhibition balance and will, therefore, differ between extraverts and introverts, brain damaged and non-brain damaged subjects, and depressant drug and stimulant drug subjects, the predictions are not precise enough. It will be necessary in order to produce more precise predictions to consider some investigations demonstrating the effects of satiation on apparent movement and to link this with Eysenck's theory.

Weiskrantz (1950) produced evidence suggesting that a stationary inspection figure can affect apparent movement to the extent of altering the length of its pathway. Inspection figures (two black rectangles) placed between the stimuli for apparent movement (two dots) resulted in an apparent lengthening of the path of the moving dot. Since by Korte's law t_{opt} varies with s (i.e. the time interval between the lights varies with the distance between them) one would predict that t would increase under Weiskrantz's conditions. Though Weiskrantz does not say anything about this, Deatherage (1954) reports a study on the effects of prolonged inspection of apparent movement. If one can consider apparent movement as resulting from a process in the cortex corresponding to the line of

movement, then, since satiation results in a functional increase in distance between the stimuli, prolonged inspection of apparent movement should lead to a functional increase in the distance between the stimuli. This by Korte's law should result in an increase in t_{opt}. This is what Deatherage found.

From this evidence we can hypothesize then that satiation or inhibition should increase t_{opt}. With regard to the upper threshold (threshold between simultaneity and movement) this means that the change from simultaneity to movement should occur at longer time intervals - the threshold should be lowered. This prediction has been based on experiments dealing with optimal movement. Because of this, and in view of the fact that the upper threshold appears to give a more clear-cut physiological threshold between optimal movement and simultaneity than the lower threshold, which is usually that between *inferred* movement and succession (Brenner, 1953), we shall concern ourselves solely with the upper threshold in the review.

Linking now this hypothesis, that inhibition results in a lowering of the threshold between simultaneity and movement, with Eysenck's theory of excitation–inhibition, we make the following predictions:

(1) Extraverts should have lower thresholds than introverts. Nicholl's findings (1955) with 27 hysterics and 26 dysthymics are in the right direction but not significant.

(2) Depressant drug subjects should have lower thresholds than stimulant drug subjects. Apart from the data to be reported here there is no evidence in relation to this prediction.

(3) That brain damaged subjects should have lower thresholds than non-brain damaged subjects. We have seen that Brenner (1953) found no differences between her brain damaged and non-brain damaged subjects under conditions producing a clear upper threshold. But Saucer and Deabler (1956) have reported that the change from movement to simultaneity occurs at longer time intervals (fewer flashes per sec) in the case of organics than in the case of lobotomized patients and normals. This is in line with our prediction.

(4) It is predicted that successive stimulation by increasing inhibition will result in a lowering of the upper threshold and that after a rest period, there will be a rise in the upper threshold once more due to the dissipation of inhibition. Since we have used a study of the effects of successive stimulation (Deatherage, 1954) to formulate our hypothesis, this may be considered a circular argument. This is true and, therefore, the data to be reviewed here may be considered as evidence defending or offending the formulation of the hypothesis in the first place. With regard to the other predictions made, of course, we do not get involved in circular arguments, and in this sense, the formulation of the hypothesis is justified.

Illig, Pflanz and von Uexkeill (1953) investigated what they called the "Moment" or "kleinste Zeitenheit" which apparently is equivalent to the upper threshold of apparent movement. They found that the greater

the number of successive estimations of the Moment that were made in one set of trials the progressively longer the Moment became, i.e. the threshold occurred at longer time intervals which is in line with Deatherage's (1954) findings. It is of interest to note here also that they found the same result with hyperventilation.

The prediction under discussion can be extended to say that any experimental conditions producing inhibition or satiation should result in a lowering of the upper threshold of apparent movement. There are a number of studies that have a bearing on this.

Deatherage and Bitterman (1952) investigated the effect of a stationary inspection figure on apparent movement. After inspection of the I-figure, a rectangle, lying in the path of the later movement, their subjects reported succession at a rate giving alternation in the pre-inspection condition. In some subjects, alternation could be reinstated by increasing the rate of alternation while for others, this produced simultaneity without intervening movement. This might appear to be contrary to our prediction that increase in satiation would result in the threshold occurring at decreased rates of alternation. But we must regard the evidence as neutral in view of Deatherage's (1954) report that the above preliminary report was incomplete since an assumption was made that if succession were observed, then obviously the rate of alternation was too slow and it was quite natural for O's to endeavour to reinstate movement by increasing the rate. That only some O's reported reinstatement appears, says Deatherage, to be an error of expectation since in his own experiment O's with opportunity given to search for the best movement up and down the alternation scale found movement at a slower rate. It should be pointed out here that Deatherage and Bitterman, and Deatherage are concerned with the simultaneity–movement threshold despite reference to succession. The succession they refer to is apparently produced at the upper threshold by satiation. The difference between the simultaneity that could be expected with a physical increase in the distance between the stimuli and the succession that results from corresponding satiational changes is noted by Deatherage, but need not concern us here.

Shapiro (1954) also used an I-figure (a circle) lying in the path of later movement and found that "the lower threshold of apparent movement was raised significantly compared with that of the control group, i.e. after inspection they required a more rapid alternation before they perceived apparent movement." McEwan (1958) in a recent extensive review of the relationships between apparent movement and figural after-effects comments that Shapiro's and Deatherage's results are in disagreement and suggests the possibility that the lack of clarity in Shapiro's experiment with regard to how the I and T figures were related may account for the discrepancy in results. His criticisms appear to be valid but will not be repeated here. More important is the fact that Shapiro was dealing with the lower threshold. He himself notes, "In evaluating these results it must be remembered that many of the subjects had difficulty in deciding whether or not they saw clear optimal movement and in fact, 7 of the 33 subjects failed to see any movement. It was decided to continue the

experiment because no reason could be seen why continuous stimulation should not have the predicted effect on any form of apparent movement. On the other hand, the possibility still exists that where object movement is clearly seen, continuous stimulation would cease to have an effect." His results, therefore, must be considered as neutral.

Brenner (1953) found that four kinds of stimulation (1) visual – fixating a circle of light, (2) auditory, a buzzer, (3) voluntary movement – pacing up and down, and (4) simple mental arithmetic, all resulted in a lowering of the upper threshold, i.e. simultaneity was perceived at longer time intervals. Though the results make an explanation in terms of direct isomorphic relations between perception and cortical events difficult, the findings with regard to the effect of visual continuous stimulation are in line with the hypothesis. Brenner also found that a rest period resulted in the threshold returning to its initial value.

Nichols (1955) also studied the effect of stimulation by an I-figure (a circle) on the upper threshold. The threshold was lowered after inspection of the circle, but the difference between the means was not statistically significant.

We may say then that the relevant evidence supports the hypothesis suggested for the effects of inhibition on the upper threshold of apparent movement.

Now we shall present an account of the drug experiments using Eysenck's drug postulate as our guide.

The drug postulate is: Stimulant drugs increase excitatory potential and decrease inhibitory potential, while depressant drugs increase excitatory potential and increase inhibitory potential.

The prediction is: The upper threshold of apparent movement will be lowered by the administration of meprobamate – a depressant drug.

THE DRUG EXPERIMENT

A small preliminary study indicated that meprobamate, as predicted, lowers the upper threshold of apparent movement (Costello, 1960a).

The procedure in the study reported here was similar in most respects to the procedure in the preliminary study.

The timer for this study was modified. The original timer had two controls, one for the time interval between the first light and the second and the other control for the time interval between the second and the first. Since it was desired in this study to keep both constant and the two dials gave some difficulty during the preliminary study, the timer was modified in order that both time intervals could be controlled by one control. This modification resulted in an alteration of the length of exposure from 45 msec to 60 msec and the time interval between stimuli now varied between 34 to 1360 msec, whereas in the preliminary study it varied from 30 to 2500 msec.

The Wither's direct exposure tachistoscope (1954) was used. The stimulus figure used was a simple one. Two circles, $^1/_2$ cm in diameter and $3^1/_4$ cm apart, were cut out of black opaque paper which was attached to a sheet

of Perspex. The Perspex was inserted into the end of the viewing tube of the Withers tachistoscope in such a way that the two bottom 5W neon lamps behind the Perspex each illuminated one of the stimulus circles. It may prevent confusion to note that the two top lamps were not used in any part of the studies reported here. The distance of the stimulus figure from the rubber eye-piece was 21 in. Further details about the tachistoscope and timer can be found in Wither's paper.

Preliminary work with the apparatus revealed the same difficulties encountered by Brenner (1953) in obtaining a satisfactory lower threshold i.e. between succession and movement, and the preliminary study dealt only with the upper threshold, i.e. between simultaneity and movement.

In this study, the upper threshold was found as in the previous study. Two ascending trials (from succession to movement) at the lower end of the scale were also run to obtain a measure of the lower threshold since it was observed that ascending trials did not seem to present as much difficulty with regard to this threshold as did descending trials (movement to sucession).

After a number of practice trials to familiarize the subject with the apparent movement phenomenon, the subject was given the following instructions: "In a few moments, I shall ask you to look into the viewing tube. You will see two lights flickering on and off simultaneously, or one light appearing to move to and fro in front of you, or two lights appearing in succession. I want you to say "Now" whenever you see a change from simultaneity to movement or from movement to simultaneity, or from succession to movement."

The time interval at which the subject said "Now" was recorded in millisec as the upper threshold. Four trials were given at the upper threshold and the limiting method was used. Ascending and descending trials were alternated with each other. The means of the four trials constituted the threshold for that session. As noted above, two ascending trials were also given at the lower threshold.

Details with regard to subjects, the times of administration of drugs and times of testing will be found in the discussion of the after-image.

Each subject was given three treatments; (1) placebo; (2) 600 mg meprobamate; and (3) 1200 mg meprobamate, on 3 different days the order of treatments being counterbalanced for subjects and sex.

Results

Table 1 shows the results of an analysis of variance carried out to test the significance of the difference between the upper threshold of apparent movement on the descending trials for the three treatments.

The data from ascending and descending trials were analysed separately since observation of the data indicated that the threshold for the two conditions behaved differently.

It will be seen from Table 1 that seven significant F ratios emerge, namely, those for "Subjects", "Treatments", "Subject/Treatment" interaction (residual 1), "Time", "Subject/Time", "Treatment/Time" interaction

and "Subject/Treatment/Time" interaction (residual 2). Fig. 1 shows the results in diagrammatic form.

Neither the day effect nor the sex effect was significant.

Looking in detail at the effects of the drug we obtain the following means:

	Mean threshold
Placebo	91·31 msec
600 mg meprobamate	101·04 msec
1200 mg meprobamate	111·32 msec

Using the *t* test of significance, we find that the only significant difference is that between the mean for placebo and the mean for the 1200 mg meprobamate day.

TABLE 1

Analysis of variance of apparent movement upper threshold on descending trials

Source	*Degree of Freedom*	*S. Squares*	*Mean Squares*	*F*	*P*
Subjects	17	620623·45	36507·26	7·62	<0·001
Days	2	15892·06	7946·03	1·66	N.S.
Treatments	2	57674·28	28837·14	6·02	<0·01
Residual (1)	32	153314·66	4791·08	17·06	<0·001
Total	53	847504·45			
Time	7	101362·99	14480·43	29·38	<0·001
Subject/Time	119	117343·28	986·08	2·00	<0·001
Day/Time	14	6774·86	483·92	—	N.S.
Treatment/Time	14	16943·81	1210·27	2·46	<0·005
Residual (2)	224	110397·00	492·84	1·75	<0·001
Total	431	1200326·39			
Final residual (3)	432	121319·50	280·83		
Total	863	1321645·89			
Between sex	1	86780·42	86780·42	2·60	N.S.
Between subjects within sex	16	533843·03	33365·19		

In order to investigate the significant "Subject/Treatment" interaction, each subject's extraversion score was correlated with his percentage drop in his upper threshold (it should be noted that though the threshold was lowered after administration of the drug and we therefore refer to percentage drop, this drop in threshold is actually represented by an increase in score, i.e. an increase in time interval between stimuli). The formulae for the four scores of change are described in the chapter on after-image. The following results were obtained:

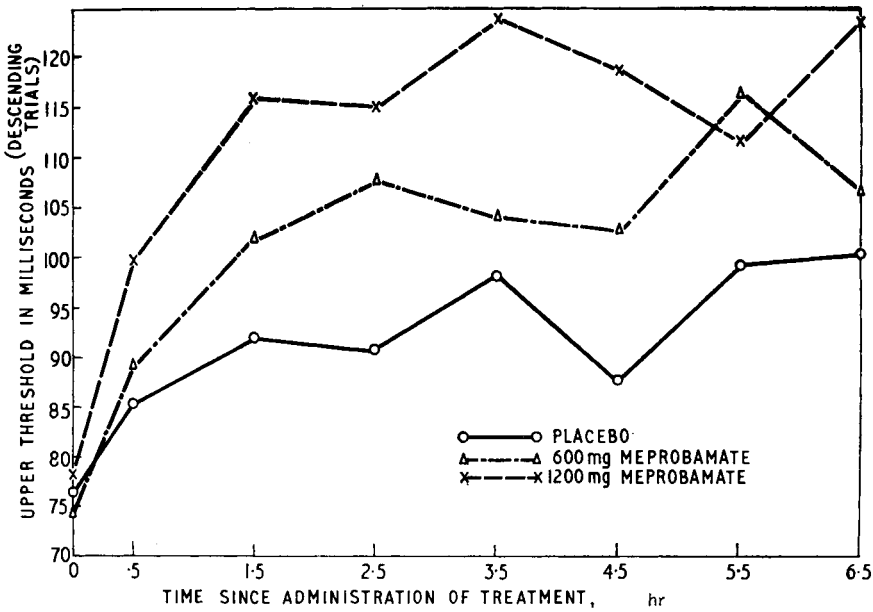


FIG. 1. The upper threshold of apparent movement (Descending trials) for the group of subjects under drug treatment.

	Extraversion
A. M. (U. T. Des) % decrease 600 mg (Max) − P(Max)	0·08
A. M. (U. T. Des) % decrease 600 mg (1 − 3) − P(1 − 3)	−0·09
A. M. (U. T. Des) % decrease 1200 mg (Max − P(Max)	−0·19
A. M. (U. T. Des) % decrease 1200 mg (1 − 3) P(1 − 3)	0·08

A negative correlation between E and the per cent change was expected in view of the results with the after-image and spiral (see relevant chapters) but none of the above correlations are significant. In our review of the apparent movement literature, we predicted a positive correlation between E and the regular apparent movement threshold (i.e. the more extraverted the lower the threshold or higher the score in terms of milliseconds). The correlation for our subjects is −0·22. This is both insignificant and in the wrong direction.

Unlike the after-image and the spiral, we did not have an unusual correlation between E and the amount of change on the apparent movement threshold to account for. It was decided, however, to correlate the amount of change with the initial score in order not to pass by possible interesting material. The results were:

	Initial
A.M. (U.T. Des) % decrease 600 mg (Max) − P(Max)	−0·30
A.M. (U.T. Des) % decrease 600 mg (1 − 3) − P(1 − 3)	−0·21
A.M. (U.T. Des) % decrease 1200 mg (Max) − P(Max)	−0·14
A.M. (U.T. Des) % decrease 1200 mg (1 − 3) − P(1 − 3)	0·14

All correlations were, of course, insignificant. Although this suggests that the satiation produced by the drug is not the same as that produced by the apparent movement stimuli, this is not reflected in a drop in reliability after drug. The test-retest reliability based on the initial scores is 0·84; based on the scores $3^1/_2$ hours after administration of placebo, 0·80; $3^1/_2$ hours after 600 mg meprobamate, 0·86; $3^1/_2$ hours after 1200 mg meprobamate, 0·94.

Let us look now at the significant "Treatment/Time" interaction.

	Means		
	Placebo	*600 mg*	*1200 mg*
Session 1	76·47 msec	74·47 msec	78·72 msec
2	85·50 msec	89·64 msec	100·36 msec
3	92·36 msec	102·69 msec	116·42 msec
4	91·47 msec	108·72 msec	115·36 msec
5	98·86 msec	104·69 msec	124·33 msec
6	88·00 msec	103·30 msec	118·83 msec
7	99·42 msec	116·97 msec	111·78 msec
8	98·39 msec	107·83 msec	124·75 msec

Applying the *t* test to the pairs of means the result is:

	Placebo—600 mg	*Placebo—1200 mg*	*600 mg—1200 mg*
Session 1	N.S.	N.S.	N.S.
2	N.S.	0·01	0·05
3	0·05	0·01	0·01
4	0·01	0·01	N.S.
5	N.S.	0·01	0·01
6	0·01	0·01	0·01
7	0·01	0·05	N.S.
8	N.S.	0·01	0·01

It is interesting to note that although the overall mean for the placebo day is not significantly different from the overall mean for the 600 mg meprobamate day, there is a significant difference $1^1/_2$, $2^1/_2$, $4^1/_2$ and $5^1/_2$ hours after administration of the treatment. Again though the overall difference between 600 mg and 1200 mg meprobamate is not significantly different, there is a significant difference $^1/_2$, $1^1/_2$, $3^1/_2$, $4^1/_2$ and $6^1/_2$ hours after administration of the treatment. It can be seen that most of the main effect is taken up by the difference between placebo and 1200 mg meprobamate where the significant difference appears $^1/_2$ hour after administration of the treatment and lasts until $6^1/_2$ hours afterwards.

Noting the significant "Subject/Treatment/Time" interaction, we would predict that the effect of the drug would occur earlier in extraverts than in introverts since, according to Eysenck's theory, satiation builds up more quickly in extraverts, one would predict, in other words, a negative correlation between E and the time to maximum effect. For 600 mg meprobamate, $r = -0{\cdot}15$; for 1200 mg, $r = -0{\cdot}08$ both, of course, insignificant.

It was predicted that the time to maximum effect would be positively correlated with weight, but the correlation for 600 mg, $r = 0{\cdot}04$ and for 1200 mg, $r = 0{\cdot}15$ are both insignificant. Correlating the four measures of change with weight, we obtained the following results:

	Weight
A. M. (U. T. Des) % decrease 600 mg (Max) − P(Max)	0·15
A. M. (U. T. Des) % decrease 600 mg (1 − 3) − P(1 − 3)	0·43
A. M. (U. T. Des) % decrease 1200 mg (Max) − P(Max)	−0·02
A. M. (U. T. Des) % decrease 1200 mg (1 − 3) − P(1 − 3)	−0·02

All the above correlations are insignificant.

Let us look now at the results for the upper threshold on the ascending trials (from movement to simultaneity). Table 2 shows the results of an analysis of variance carried out to test the significance of the difference between the upper threshold of apparent movement on the ascending trials for the three treatments.

It will be seen from Table 2 that eight significant F ratios emerge, namely, those for "Subjects", "Treatments", "Subject/Treatment" interaction (residual 1), "Time", "Subject/Time" interaction, "Treatment/Time" interaction, "Subject/Treatment/Time" interaction (residual 2), and for "Sex". The results are presented in diagrammatic form in Fig. 2.

The "Day" effect and "Day/Time" interaction were not significant.

TABLE 2

Analysis of variance of apparent movement upper threshold on ascending trials

Source	*Degree of Freedom*	*S. Squares*	*Mean Squares*	*F*	*P*
Subjects	17	892142·51	52478·97	28·95	<0·001
Days	2	11197·27	5598·63	3·09	N.S.
Treatments	2	48801·87	24400·93	13·46	<0·001
Residual (1)	32	58001·11	1812·53	10·00	<0·001
Total	53	1010142·76			
Time	7	13004·98	1857·85	6·86	<0·001
Subject/Time	119	44019·37	369·91	1·37	<0·01
Day/Time	14	4078·97	291·35	1·08	N.S.

Source	*Degree of Freedom*	*St Squares*	*Mean Squares*	*F*	*P*
Treatment/Time	14	8172·60	583·76	2·16	<0·01
Residual (2)	224	60638·52	207·71	1·49	<0·01
Total	431	1140057·20			
Final Residual (3)	432	78259·50	181·16		
Total	863	1218316·70			
Between sex	1	195993·34	1959931·34	4·50	<0·05
Between subjects within sex	16	696149·17	43509·32		

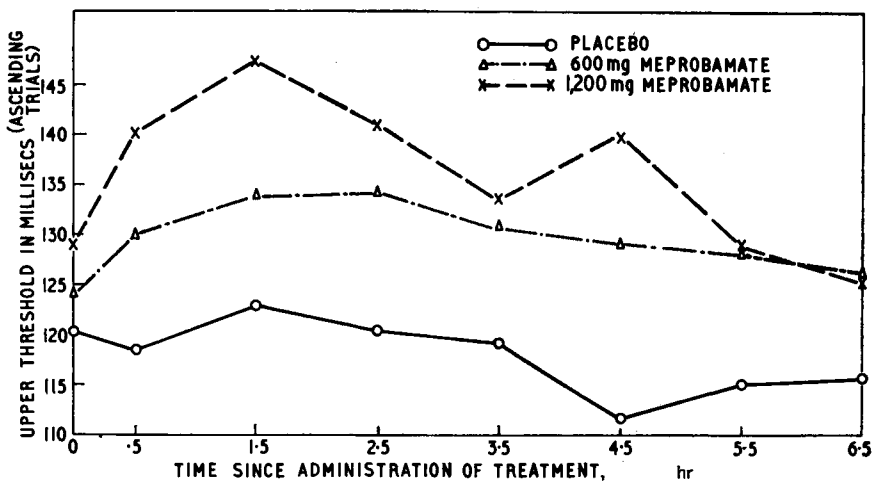


FIG. 2. The upper threshold of apparent movement (ascending trials) for the group of subjects under drug treatment.

Looking in detail at the main drug effect we obtain the following means:

	Mean upper threshold (ascending)
Placebo	117·77 msec
600 mg meprobamate	129·88 msec
1200 mg meprobamate	135·83 msec

Using the '*t*' test of significance, we find that the mean for the placebo is significantly different from both the mean for the 600 mg day and the mean for the 1200 mg day. The mean for the 600 mg day is not significantly different from that for the 1200 mg day.

Correlating E with the four measures of change produced the following results:

	Extraversion
A. M. (U. T. Asc.) % decrease 600 mg (Max) − P(Max)	−0·13
A. M. (U. T. Asc.) % decrease 600 mg (1 − 3) − P(1 − 3)	−0·13
A. M. (U. T. Asc.) % decrease 1200 mg (Max) − P(Max)	0·01
A. M. (U. T. Asc.) % decrease 1200 mg (1 − 3) − P(1 − 3)	0·00

The correlation between E and the apparent movement upper threshold (ascending trials) for the first two trials is 0·11 which is both insignificant and in the wrong direction since we predicted a positive correlation between E and the upper threshold.

Correlating the measures of change with the initial score produced the following results:

	Initial
A.M. (U.T. Asc.) % decrease 600 mg (Max) − P(Max)	−0·09
A.M. (U.T. Asc.) % decrease 600 mg (1 − 3) − P(1 − 3)	−0·18
A.M. (U.T. Asc.) % decrease 1200 mg (Max) − P(Max)	−0·14
A.M. (U.T. Asc.) % decrease 1200 mg (1 − 3) − P(1 − 3)	−0·35

Once again it would seem that the processes produced by the drug are in some way different to those produced by stimulation with the apparent movement stimuli though the reliabilities do not change significantly. The test-retest reliability based on initial scores was 0·96; based on scores $3^1/_2$ hours after administration of placebo, 0·92; $3^1/_2$ hours after 600 mg meprobamate, 0·84; $3^1/_2$ hours after 1200 mg meprobamate, 0·96.

A breakdown of the significant "Treatment/Time" interaction produces the following results:

	Means		
	placebo	*600 mg*	*1200 mg*
Session 1	120·28 msec	124·36 msec	128·33 msec
2	118·72 msec	130·14 msec	140·47 msec
3	123·05 msec	134·19 msec	147·53 msec
4	120·42 msec	134·47 msec	141·44 msec
5	119·11 msec	130·78 msec	133·83 msec
6	111·78 msec	129·53 msec	140·17 msec
7	115·03 msec	128·89 msec	128·97 msec
8	115·80 msec	126·67 msec	125·89 msec

Applying the '*t*' test to the pairs of means the result is:

	Placebo – 600 mg	*Placebo – 1200 mg*	*600 mg–1200 mg*
Session 1	N.S.	0·05	N.S.
2	0·01	0·01	0·01
3	0·01	0·01	0·01
4	0·01	0·01	N.S.
5	0·01	0·01	N.S.
6	0·01	0·01	0·01
7	0·01	0·01	N.S.
8	0·01	0·01	N.S.

It can be seen that administration of 600 mg meprobamate produces a significant difference from the placebo score $^1/_2$ hour after treatment and the effect lasts till $6^1/_2$ hours after treatment. Administration of 1200 mg meprobamate produces the same result when we compare the scores with the placebo scores. Comparing 600 mg with 1200 mg, we find that there is a significant difference $^1/_2$ hour and $1^1/_2$ hours after treatment and again at $4^1/_2$ hours after treatment.

As with the descending trial, one would predict a significant negative correlation between E and the time to maximum effect. For 600 mg, r = = 0·12; for 1200 mg, r = – 0·12 both are insignificant.

The prediction that there would be a positive correlation between time to maximum effect and weight was not borne out. For 600 mg, r = 0·442; for 1200 mg, r = –0·29. Indeed one might say that they were dangerously in the wrong direction. Correlating the four measures of change with weight produced the following insignificant results:

	Weight
A. M. (U. T. Asc.) % decrease 600 mg (Max) – P(Max)	–0·03
A. M. (U. T. Asc.) % decrease 600 mg (1 – 3) – P(1 – 3)	–0·11
A. M. (U. T. Asc.) % decrease 1200 mg (Max) – P(Max)	–0·04
A. M. (U. T. Asc.) % decrease 1200 mg (1 – 3) – P(1 – 3)	0·10

That the determination of the upper threshold of apparent movement involves different factors on the ascending trials as compared with descending trials is indicated by the low correlation between the initial scores for ascending trials and the initial scores for descending trials, r = 0·32. It can be seen also in the correlations between the four measures of change with drug on ascending trials and the same four measures on descending trials.

	A.M. (U.T. Des) % decrease
A. M. (U. T. Asc.) % decrease 600 mg (Max) – P(Max)	0·08
A. M. (U. T. Asc.) % decrease 600 mg (1 – 3) – P(1 – 3)	–0·03
A. M. (U. T. Asc.) % decrease 1200 mg (Max) – P(Max)	0·27
A. M. (U. T. Asc.) % decrease 1200 mg (1 – 3) – P(1 – 3) –	–0·09

Fig. 3 shows the results for the lower threshold (succession-movement) in diagrammatic form. It can be seen that the drug did not have any systematic effect on this threshold and a statistical analysis of the data was not carried out.

Correlations between the scores on the apparent movement test and those on the after-image and spiral test were insignificant. A discussion of this lack of correlation between the tests will be found in the chapter on the after-image.

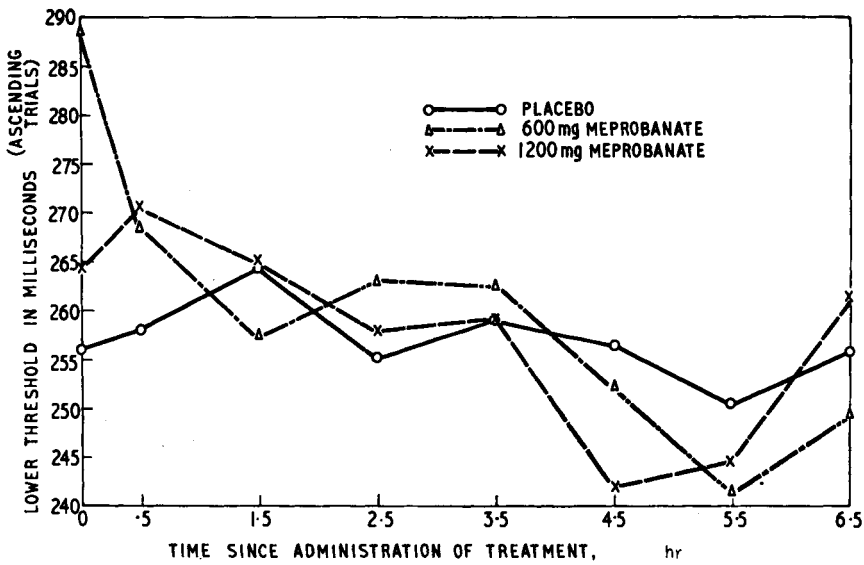


FIG. 3. The lower threshold of apparent movement for the group of subjects under drug treatment (Study II).

Summary and Conclusions

(1) A distinction must be made between the upper and the lower thresholds of apparent movement. A review of the literature and pilot work indicated that only the upper threshold (between simultaneity and movement) could be considered a true psycho-physiological threshold. It is to the upper threshold that we refer when we talk below about apparent movement in discussion of the predictions.

(2) A review of the literature on apparent movement indicated that we could only tentatively accept that apparent movement had important central components. It also indicated that the data available was in line with the predictions from Eysenck's theory:

(a) That extraverts should have lower thresholds than introverts. Significant correlations between extraversion and the apparent movement thres-

hold were not found in this investigation due to the smallness and homogeneity of the groups.

(b) That successive stimulation or any experimental situation producing an increase in inhibition (e.g. the use of I-figures) will result in a lowering of the threshold of apparent movement.

(3) The data with regard to the prediction that brain damaged subjects should have lower thresholds than normals is conflicting.

(4) There is nothing in the literature with regard to the effects of a depressant drug on apparent movement, but the study reported here confirms the prediction, based on Eysenck's theory, that a depressant drug would lower the threshold.

It was found necessary to present the results for descending trials and ascending trials separately since there were some differences between the thresholds obtained by the two methods.

(5) As predicted, meprobamate did not have any systematic effect on the lower threshold of apparent movement.

Chapter 11

DEPRESSANT-STIMULANT DRUGS, INHIBITION AND THE VISUAL CONSTANCIES

JOHN SYLVESTER*

Introduction

Some writers have tended to associate Eysenck's excitation–inhibition theory with the Kohler–Wallach theory of "satiation". Thus both Holland and Costello regard "excitatory potential" as a diffused cortical process, more or less electrical in nature, and both use the word "satiation" interchangeably with "inhibition", in certain contexts. Eysenck himself, however, draws a distinction between "temporal" and "spatial" inhibition (Eysenck 1957), after Pavlov's "internal" and "external" inhibition, and likens them to the concepts of fatigue and distraction respectively. When discussing perception, it is true, he believes that temporal and spatial inhibition "go hand in hand", but he insists he uses temporal inhibition as a "molar construct" only (*ibid.* p. 190) and elsewhere allows that spatial inhibition is simply an intervening variable. As such, it is certainly subject to explanations other than the Kohler physiological isomomorphism. Pavlov clearly regarded inhibition as something much more than a kind of fatiguing process in the brain. He says: "In the highest part of the central nervous system, in which there is a constant collision of innumerable influences from the external world, it is comprehensible that among the different conditioned reflexes there is an incessant struggle, a choice among them at any given moment. Consequently there are constantly arising cases of inhibition among these reflexes." (Pavlov–Anrep, 1927).

Now, as will be more fully discussed in the section on apparent movement, the Kohler–Wallach satiation theory cannot be regarded as entirely satisfactory and it may, therefore, be useful to consider another model, one which will take into account the fact that so much of both behaviour and perception is *learned* and/or influenced by previously-acquired drives, attitudes and sets. Confining ourselves to perception, let us consider Pavlov's notion of the 'analysers', bringing it into contemporary fashion by using the language of the digital computer.

In the first place, it must be re-emphasized that perception is not just a matter of receiving and being aware of a stimulus (except in certain laboratory situations); it involves interpretation of stimulus-patterns and it proceeds by the utilization of cues. Perception in ordinary life involves

* The writer is indebted to the Wallace Laboratories for support of this research.

the notion of *meaning*, which involves memory, which involves previous experience, and also it involves calculation.

The constancy phenomena clearly demonstrate the amount of calculation used in perception. Size-constancy, for example, is explained by the fact that the observer takes into account the distance away when perceiving any object. But in order to take distance into account, it is first necessary to estimate what the distance is. This is done by using the cues of convergence, accommodation, retinal disparity, linear perspective, aerial perspective, masking, monocular movement parallax, and so on. The data gained from these cues must then be used in a complex calculation to arrive at an estimation of the distance. All this process takes place very rapidly and usually quite unconsciously. It is what was described by Helmholtz as "unconscious inference," a term which has been much attacked by the Gestaltists and others. Certainly, if it refers to some uninvestigateable mystery of the "Unconscious," in a Freudian sense, the critics would seem justified. On the other hand, if the process intervening between raw stimulus-pattern and the attaining of a *percept*, – complete with constancy, meaning and learned response, – is seen as effected by some sort of computing mechanism, then it is a different matter. A "perceptual computer" explains many of the facts of complex perception and is a hypothesis open to investigation. The "programming" of such a mechanism is a matter of learning, of previous experience. Individual differences may be seen as resulting from some interaction between differences of construction and operation of the computer and differences of "programming" – i.e. anatomical – physiological differences and differences of experience plus drives, sets, attitudes etc.

There are obviously two main lines of approach to the exploration of the nature of such a mechanism. One can manipulate the data fed into it, as with the Ames demonstrations, for example, or such experiments as the size–weight illusion, or one can attempt to manipulate the physiology or anatomy, as in the case of the work of Lashley (1951), or Werner and Thuma (1942).

Another obvious method in this latter area is to use drugs. The depressant drugs such as alcohol or the barbiturates tend to reduce the efficiency of the organism; they knock out some cells and hence raise reaction-times and, in general, tend to inhibit cortical pathways. Using the concept of an "analyser", then, we can predict that a depressant drug should lessen its efficiency, making it less able to use all the data available and hence more likely to end up with (predictable) wrong answers. This is only a more detailed formulation of Eysenck's drug postulate that "depressant drugs increase inhibitory potential" and the four experiments to be described are meant to test such a hypothesis. It may be supposed, of course, that stimulant drugs will have a reverse effect to depressant ones.

There is one further point to be clarified. The word "cue" refers to available information in the external environment in the forms of such physical energies as light waves, air-pressure patterns etc. Now between the light ray which reaches the eye and the pulses in the optic nerve there is a change of energy-form and also a considerable loss of data (as may be readily demonstrated, for example, with a magnifying glass or a microscope).

Hence there is a considerable difference between the signals reaching the eye and the signals reaching the brain. I shall term the latter *physiological cues* (after Hansel, 1953).

Experiments using stimulant and depressive drugs on size constancy, shape constancy and apparent movement will now be described.

Size Constancy

Since the estimation of the real size of an object seen from a distance depends on the use of cues, it follows that when no cues are available the perceived size will approach that predictable from the law of visual angle, or retinal image. This was demonstrated by Holway and Boring (1941) who further showed that as cues are successively added the perceived size gets nearer and nearer constancy i.e. real size.

Now, clearly, for correct estimation, cues have not only to be available, they then have to be made use of. If depressant drugs increase inhibition, or, as I have put it, lower the efficiency of the visual analyser or "computer," then they should have the same effect as cue-reduction. If a subject is required to estimate the size of an object in conditions where only a few cues are available, one would except him to be nearer the retinal-image size (i.e. show less constancy) when under a depressant drug than when not drugged. Conversely, a stimulant drug which facilitates cortical pathways, would be expected to produce estimations near the real size even under conditions of partical cue-removal. This is the hypothesis to be tested.

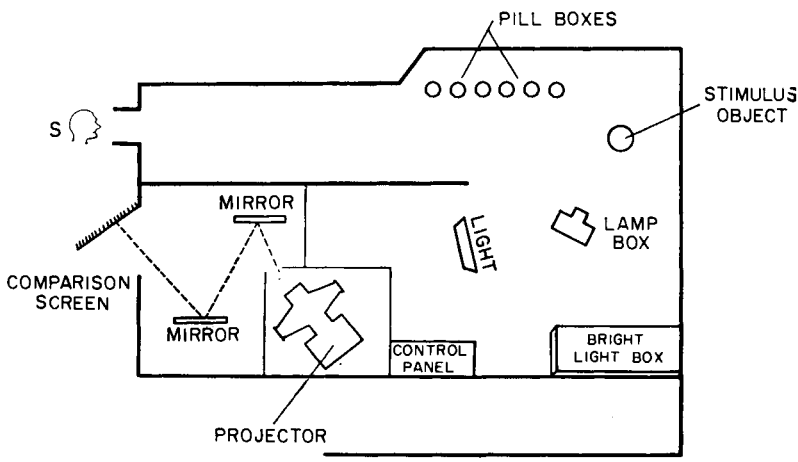


FIG. 1. Schematic diagram of apparatus used in the constancy experiments.

Method

White cardboard disks were presented to subjects at a distance of 9 ft under three conditions: (1) monocularly, with very poor illumination and with linear-perspective cues largely removed; (b) binocularly under the same conditions, and (c) binocularly with full light and cues.

Seven cards were used, of diameters 2, 4, 6, 8, 11 and 14 cm. Twenty centimetres from the subject a screen was placed upon which could be projected varying circles of light from behind. Subjects were required to estimate the real size of the cards by instructing the experimenter to alter the size of the light-circle on the screen to equality.

A diagram of the apparatus is given in Fig. 1. Two thirds of the way down one half of a long bench in a dark room a viewing-tunnel was constructed from "handy-angle" metal strips, covered with black velvet-finish paper. The other half of the bench, at the end where the subject sat, accommodated the comparison-screen and a film-strip projector giving a back-projection with the aid of mirrors. At the far end of this side of the bench was a glass-fronted box housing a fluorescent lamp and two 100 watt tungsten light-bulbs. A further light was provided at the centre of the set-up and a small lamp-box illuminated the disks.

Under the "dark" conditions only the lamp-box and the projector were switched on (a small amount of stray light escaped from the projector). Under the "full light" condition all lights and lamps were on, including the room lights. Also, with this condition, a row of white 2 in. pill-boxes was placed in a line away from the stimulus-object, in order to provide perspective cues.

Nine subjects were used, six in a balanced incomplete block plus three replications; Dexedrine (10 mg) was used as the stimulant drug, Sodium Amytal (227 mg) as the depressant and a placebo was used as a control condition. Subjects were all unpractised, they were females aged 19—27.

TABLE 1

Estimation of disk-size (in millimeters) under different drug and cue-reduction conditions

Subject	*Dexedrine*			*Amytal*			*Placebo*		
	Monoc.	*Binoc.*	*Full Light*	*Monoc.*	*Binoc.*	*Full Light*	*Monoc.*	*Binoc.*	*Full Light*
A	82·6	87·3	90·0	69·7	77·6	86·0	97·7	95·0	90·0
B	78·9	84·0	86·4	73·0	83·1	89·6	77·1	83·7	85·0
C	100·4	105·9	102·0	76·3	84·2	92·9	91·0	100·1	97·7
D	76·7	77·1	77·5	59·3	57·6	67·0	50·4	58·9	61·3
E	54·5	77·6	93·1	31·6	81·7	99·0	45·6	78·4	86·7
F	88·9	81·7	88·0	81·6	89·7	103·4	60·0	86·9	87·0
G	70·6	80·9	86·0	74·0	84·9	84·0	78·1	80·0	87·1
H	79·7	90·4	97·0	76·1	78·0	85·0	83·6	79·7	85·0
I	63·6	63·6	87·1	27·0	76·6	74·1	41·9	67·6	79·1
MEAN	77·3	83·2	89·7	63·2	79·3	86·8	69·5	81·1	84·3

Results

As can be seen from Table 1 and Fig. 2, although there are large individual differences, the depressant drug sodium Amytal had the effect of considerably reducing the estimate – i.e. making it approach retinal-image size – under conditions of cue-reduction (this under maximum cue-reduction, failed in only one case (subject G) and there only by an amount small enough to be regarded as experimental error). The differences of estimates in the monocular/dark condition between Dexedrine and Sodium Amytal were significant at the 0·01 level, those in the binocular/dark condition significant at 0·05.

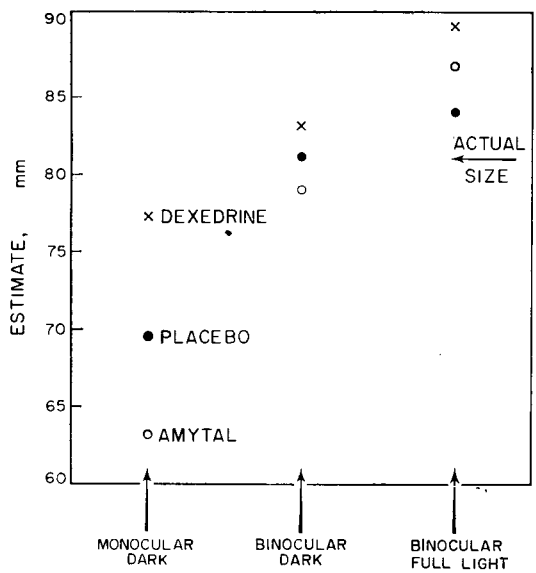


FIG. 2. Diagram showing effect of the drugs on subjects' estimates of size under the different experimental conditions.

Differences between the placebo condition and the two drugs were not significant.

With regard to the fact that the estimates with full cues and, with Dexedrine, binocularly in reduced illumination, are *larger* than the actual size (i.e. a Brunswik ratio greater than 1), it should be pointed out that the comparison light was brighter than the cards and Hsia (1943) showed that difference of intensity between stimulus– and comparison–objects always increases constancy. Also Brunswik (1940) has data to show that absolute stimulus difference affects constancy in many ways — distance apart, visual angle, etc. None of these things were controlled in this experiment because we were concerned only with *comparative* data — the differences between depressant and stimulant drugs rather than with absolute values which would need extremely complex experiments to determine.

While it is clear that the results of this experiment give support to the prediction that depressant drugs reduce constancy, another point of interest and perhaps importance arises, namely, to what extent is a depressant drug affecting the operation of the central cortical computor, as distinct from it merely reducing the data fed into this by reducing the *physiological* cues? The drug may, that is, be working only on the afferent pathways or at the receptor ends of these (physiological evidence suggests that both Dexedrine and Sodium Amytal work via the "arousal mechanism" of the reticular system, thus affecting the ability of the central – cortical – mechanisms to accept data offered to it).

A study of *shape* constancy might provide one way of exploring this question. Whereas size-constancy depends on cues physical, shape constancy appears to be a matter of previous experience, i.e. shape constancy depends not so much on the adequacy of the fed into the analyser, but on how the analyser has been "programmed" — on its "store", so to speak. Accordingly, a further experiment was carried out and this will now be described.

TABLE 2

The shape-constancy experiment: mean estimates of width of rectangles (in centimetres)

Subject	*Dexedrine*			*Amytal*		
	45°	*60°*	*72°*	*45°*	*60°*	*72°*
1	9·3	9·2	8·2	10·8	9·1	6·9
2	10·2	10·1	10·3	11·4	10·4	9·9
3	8·3	7·2	6·9	8·2	7·2	6·3
4	9·8	9·6	9·1	9·3	8·6	7·4
5	10·0	8·2	7·8	9·5	8·9	6·0
6	9·2	8·8	8·1	10·1	7·0	7·5
7	8·3	7·0	7·1	8·1	7·5	5·8
8	9·4	9·4	9·6	9·7	5·7	5·9
9	9·2	9·0	9·3	8·3	6·2	6·1
Mean	9·3	8·7	8·5	9·5	7·8	6·9

Shape-constancy

Although Thouless (1931a) obtained estimations veering toward the expectation from the visual image law even without cue reduction, it may be thought that these were mostly the result of the ambiguity of his instructions to the subjects (the importance of this point is demonstrated by Klimpfinger (1933)). In some preliminary trials in the experiment to be described it was found that with full cues present and with firm instructions to estimate the *actual, real* width of the stimulus-card, full constancy was maintained

up to angles of over 80°. Consequently it was decided to reduce cues to a certain extent, as in a later experiment by Thouless (1931b).

Method

Substantially the same apparatus as in the previous experiment was used. Instead of disks, however, white rectangular cards were used, being set upon a turntable operated by a remote control servo-motor with a graduated scale so that the cards could be presented at varying angles to the subjects. The cards were rotated about the *vertical* axis i.e. the retinal image of the horizontal axis, or *width* of the cards, would diminish as the angle of inclination away from the subject (the angle between the card and the line drawn at right angles to the line of sight) increased. Five cards were used, all 15 cm high and of widths of 5, 7·5, 9, 12 and 15 cm. On the comparison-screen, a rectangle of light was projected. The height of this remained always at 15 cm but its width could be continuously varied by the experimenter. Subjects were required to instruct the experimenter to adjust the width of the light-rectangle until it indicated their estimate of the *actual* width of the stimulus-card. Conditions were as in condition 2 of the size-constancy experiment i.e. binocular viewing of a poorly illuminated card in a room darkened but with some stray light. Nine subjects, female, aged 19–30, were used, each receiving 10 mg Dexedrine, 227 mg of Amytal and a placebo. Stimulus-card and angle of presentation were randomized. Three angles of presentation were used: 45°, 60° and 72°.

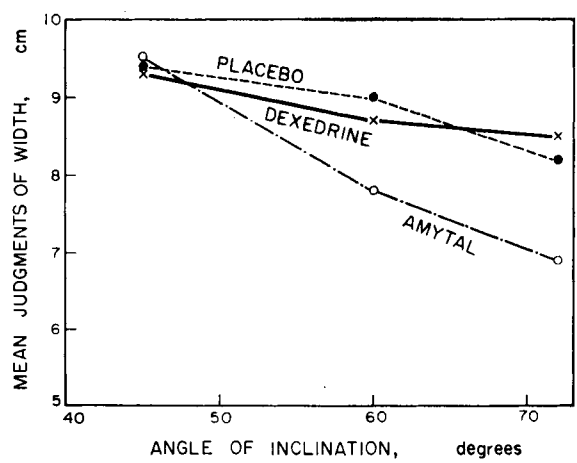


FIG. 3. Diagram showing subjects' estimates of shape (i. e. width–height ratio) at different angles of presentation and the effects of the drugs.

Results

The means of each subject's five readings are given in Table 2. It will be seen from this and from Fig. 3 that constancy was considerably diminished by Sodium Amytal at 60° and 72°. At these angles, subjects under Dexedrine

also fell short of compete constancy, but not by nearly so much. The difference of estimates between the Dexedrine and Amytal conditions at 72° is significant at the 0·01 level. At the 45° angle of presentation, there is a reversal of this trend, but it is small and not significant.

Results of the placebo condition are given in Table 3. The variance between individuals here is larger than under either drug condition and at 60° the mean is nearer constancy than with either Amytal or Dexedrine. This point will be discussed later.

TABLE 3

Mean estimates of width of rectangles (in centimetres). Placebo condition

Subject	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*Mean*
45°	9·2	10·2	8·3	9·7	11·2	9·3	8·0	9·4	9·5	9·4
60°	7·6	12·2	7·9	9·9	11·0	8·4	7·1	9·8	7·2	9·0
72°	6·0	11·4	9·1	8·7	10·0	8·2	5·4	7·4	7·5	8·2

Although the results of this experiment clearly show an inhibition of shape-constancy with a depressant drug, they cannot be said to settle the question of whether the inhibition is of the central analyser or of physiological cue, since, of course, *some* cue to angle of inclination is necessary and was present in the experiment. Stavrianos (1945) claimed that "changes in the accuracy with which inclination is judged will be accompanied by changes in the accuracy of shape perception such that, when the inclination of an object is accurately perceived, its apparent shape will coincide with the actual shape; when inclination is underestimated ... the apparent shape will deviate from the actual in the direction of ... overconstancy." but this is not supported even by his own data and the same applies to Koffka (1935) who had earlier made a similar pronouncement. Discussing this point, Graham (1951) concludes there is no evidence for it. It will be seen from Fig. 4 that all the estimations, at any inclination, are well away from the judgments to be expected on the visual angle law, but that the depressant drug curve most nearly approaches this. The Dexedrine curve seems to flatten out at 60°.

The fact there are no significant differences between the Amytal, Dexedrine and placebo conditions at the 45° angle of inclination may perhaps be put forward in support of the cue-reduction hypothesis, but as the curves cannot be continued below 45° in the absence of the necessary data, this is of doubtful significance.

Evidently what is wanted to settle this issue is some kind of constancy — some kind of higher-order perceptual process — which is in no way affected by compex patterns of cues. It will be urged in the next section that there is such a phenomenon in *apparent movement* or, at any rate, in a certain

aspect of it, namely, *the bottom threshold* in experiments where only the time-interval is varied (where Costello goes wrong in dismissing this threshold will be explained in the next section).

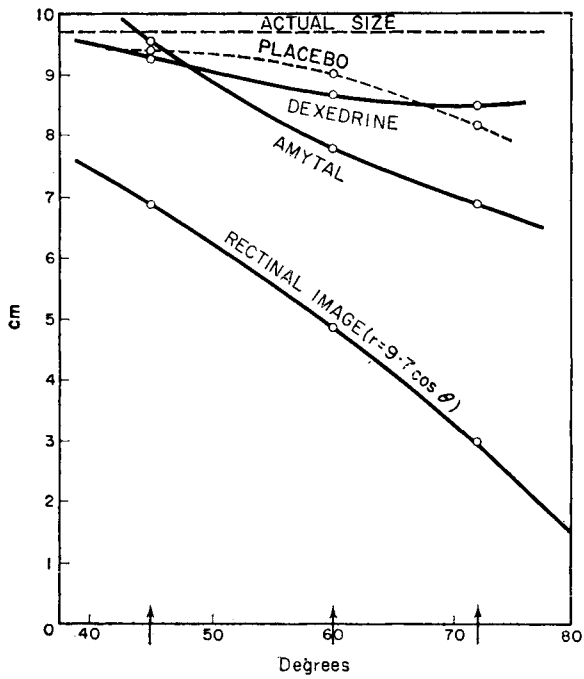


FIG. 4. Graph of width expected to be seen according to the visual angle law (shape of retinal image) compared with results actully obtained under different drug conditions

Apparent Movement

There has been controversy over the cause of stroboscopic visual apparent movement for many years, it being held, on the one hand, that the explanation is physiological or "structural" and, on the other, that it is, at any rate primarily, a matter of previous experience of real movement, i.e. a function of learning.

Wertheimer (1912) postulated diffusion circles of excitation (in a homogenous cortex) which, as they expanded towards each other from two points of stimulation would produce "short-circuiting". This hypothesis presents difficulties *a priori*, for it would seem to follow that (a) a single light-flash should appear to expand during the post-stimulation persistence period, and (b) two steady lights should appear as a *bar* of light, neither of which, of course, occurs. Also, it would hardly seem to fit the known punctate nature of the brain. However, the Kohler–Wallach theory of "satiation"

takes this notion as its starting-point and a number of writers have urged this type of theory as the explanation of apparent movement, among whom may be mentioned Kohler (1940), Deatherage and Bitterman (1952), Shapiro (1954), Saucer (1954), McEwen (1958), and Costello (1962).

Apparent movement is commonly subsumed under the general heading of Figural After-Effects and perhaps the majority of workers in that field have embraced the satiation theory. Objections to this theory, nevertheless, are serious and persistent. Hebb (1949) has pointed out that a quantity of neurological and neurophysiological data are at variance with the theory and Eccles (1958), for example, has shown that the inter-neurone medium has an electrical resistance which is negligible, that voltage can only be developed along the neurones themselves, that dendrites are *generators* of voltage pulses and that any neurone excitation which might be generated by electro-magnetic fields could only have nuisance value. The hypothesis requires that the figure-field includes both hemispheres of the cortex, but Curtis (1940), and Von Borin *et al.* (1942) have indicated that the corpus callosum does not act as a conducting medium between the two parts of the visual field and that since the characteristics of the intercellular fluid varies from time to time, distortion would be expected to occur, on satiation theory, where in fact in does not. Lashley *et al.* (1951), and Sperry *et al.* (1955) have subjected the satiation hypothesis to experimental tests, the former by short-circuiting the visual cortex and the cortically represented macular region of workings by gold leaf and gold pins, the latter by planting mica plates and tantalum wires in the cortex and underlying white matter of cats. No distortions of visual discrimination were obtained by either group of workers. Kohler and Wallace themselves, (1944) for example, as well as Walthall (1949), and Marks (1949) have reported that after prolonged inspection of a closed figure, it appears smaller, more distant, desaturated and, in some cases, disappears altogether, but according to the theory it should appear larger — indeed Kohler and Fishback (1950) use the argument of excess of satiation within boundaries to account for the disappearance of the Muller–Lyer illusion with repeated exposures. Culbert (1954) and others have shown that curved lines appear straighter regardless of their relation to previously inspected lines of greater or lesser curvature, and this would seem to be quite irreconcilable with satiation.

Certainly many of the phenomenological findings of figural after-effects can be explained as well, if not better, by other theories, notably by the "adaptation" hypothesis tentatively suggested by Gibson (1931) (who made the first major investigations of the phenomena) and subsequently developed by Fox (1951), Witkin (1954), and Helson (1948). Both the well-known fact that organisms tend to react to *change* of stimulus pattern and the work on the "orienting reflex" (Sokolov, 1955) have been ignored by investigators working with "satiation" hypotheses.

In general, it would seem that a single molecular explanation of figural after-effects and related phenomena is improbable. Apparent movement, especially, appears open to several explanations which will not fit with at least some of the phenomena covered by the title Figural After-Effects

and it would seem advisable, when investigating phenomenal movement, to treat it as quite separate.

Opponents of physiological theories of apparent movement include De Silva (1926), Linke (1929), Higginson (1926), Neuhaus (1930), Peterman (1932), Graham (1951), Pieron (1952), Hansel (1953), Jones and Bruner (1954), Toch and Ittelson (1956), and Sylvester (1960). In one way or another, these all point out that apparent movement is subject to attitudinal factors and, therefore, cannot be an elementary physiological product; they suppose instead either that the retinal-cortical stimulation patterns produced by an apparent-movement situation are indiscriminable from those produced in the perception of real movement, or else that apparent movement is mostly a matter of a central interpretive mechanism which is largely influenced by past experience. As Hansel (1953) has pointed out, "if two different stimuli will, under certain conditions of stimulation, produce equivalent sets of physiological cues, it may be deduced that there will be no difference in the information by which the two sets of cues may be differentiated and perception will be in terms of the manner in which this particular set of cues reacts with past experiences." (The Ames illusions are striking examples of this.) Because (1) anticipatory fixations are the common method of eye-movements in the perception of moving objects, and (2) the establishment of a percept takes time and involves refractory phases, it may be supposed that during perception of real movement, the physiological cues reaching the cortex are intermittent and, therefore, not different from the stroboscopic presentation of stationary stimuli. Since the subject has greatly overlearned to perceive successive stimulations of different parts of the retina, at certain speeds, as movement, he *must* see movement in the apparent-movement situation.

And many situations can be a great deal more sophisticated than this. It is notorious that apparent movement is much more readily perceived in Piccadilly Circus or Times Square than it is in the laboratory and it seems clear that the reason for this is that there is more *meaning* in advertisement displays than there is in the standard two-light laboratory situation. A good example of an experimental demonstration of this point has been provided by Toch and Ittelson (1956) who exposed a single stimulus-figure followed by two similar figures outside it, one above and one below. When the figures were shaped like aeroplanes, upward movement was perceived; when shaped like bombs, downward movement was seen. It is difficult to see how this result could be explained by a "physiological" theory.

However, it is perhaps misleading to talk of "physiological" and "non-physiological" theories, for, of course, supporters of adaptation or learning theories are not attempting to deny that brain and retinal physiology is relevant; it would be better to differentiate the views by describing them as, say, the "simple" and the "compex" theories. The Wallach–Kohler theory ascribes the basis of figural after-effects and apparent movement to postulated events in the visual cortex only; adaptation and learning theories insist that these phenomena are crucially affected by events occurring over a much wider area of the central nervous system, particularly in "association"

or "interpretation" fields — the computer again, the "store" of which is formed by learning processes.

But let us consider the phenomenon itself and its measurement. Apparent movement occurs under certain conditions and not under others, the most important variables being the distance apart of two or more flashing lights, their duration and the time interval between them. A typical experimental set-up is to have two lights at a fixed distance apart and to vary the time interval between flashes. When the time interval is very long the lights are seen as flashing one after the other, in succession. As it is shortened, there occurs a point when what is seen is one light moving. This point is known as the bottom threshold, the top threshold referring to the interval at which the perception of movement changes to seeing both lights on at once — simultaneity.

Now Costello specifically ignores the bottom threshold, but is this not a case of throwing out the baby with the bath-water? Apparent movement at the bottom threshold may well *be* a matter of "inference", but this would make it more, not less, of a psychological fact. It would seem, indeed, to be very much related to constancy. The top threshold, clearly, is a different matter. This is dependent on the speed of energy-charge at the periphery and the speed of nerve-conduction plus the duration of stimulus-trace. It is much more purely physiological — a matter of what physiological cues reach the central areas. We can say then that if a drug affects the top threshold, much of its effect must be on the peripheral receptors and afferent pathways, while if it effects the bottom threshold, its effect is on central mechanisms. To manipulate the top threshold is to manipulate the data fed into the analyser, to manipulate the bottom one is to affect the workings of the 'computer' itself.

Predictions concerning the upper threshold, then, can be made along the lines of the 'physiological' theories and will be the same as Costello's, namely, that a depressant drug will produce inhibition of afferent pathways and lower the threshold.

To make predictions about the bottom threshold, however, needs some further explanation. No-one doubts that the *purpose* of perception is biological adaptation. For successful adaptation it is necessary to have correct information about the environment and as many writers have pointed out (see Osgood, Chap. 7, 1953), the contrast and constancy mechanisms work to this end — work to perceiving the world as it is, which is to say, perceiving it against the totality of previous experience. Now, it is part of our previous experience that if an object disappears from view and later reappears in a different part of the visual field, then that object has moved (or we have). All our experience is of objects which persist in time, though they may not be always in view. We are continuously interpreting disappearance-appearance in terms of movement and obviously have a drive so to do whenever possible (this is more fully discussed later). It would, therefore, follow that if the inference mechanism is working at optimum efficiency, we would see movement with fewer movement-cues than if it were not — just as with the constancies. Further, there would be a greater range of conditions over which apparent movement would be seen. Hence one can

predict that a depressant drug, tending to inhibit the operation of the computor, will raise the bottom threshold of apparent movement, while a stimulant drug will lower it.

Some workers have experienced difficulty with the measurement of the bottom threshold, it is true. This would seem to be due to two factors, the first of which is a certain ambiguity in the subjects' understanding of what is wanted — a confusion or uncertainty between "*phi*" movement and "*beta*" movement. "*Phi*" movement is merely an *impression* of something having moved or "movement that does not involve object movement" (Wertheimer, 1912). With "*beta*" movement, or *optimal* movement as it is alternatively called, the subject sees or thinks he sees an object (a light or a figure) actually moving. This is the phenomenon on which motion-pictures are based. "*Phi*" movement occurs between the threshold for optimal *(beta)* movement and succession and hence it is obviously important that subjects should be quite clear about what it is they are being asked to do or, better still, subjects should not be given the responsibility for making decisions here.

The second factor contributing to difficulty in measuring the bottom threshold is the fact that on *downward* runs (i.e. lengthening of the time-interval between lights) the effect of "set" is strong enough to cause the subject, particularly an unsophisticated one, to continue to report movement over very long intervals, sometimes, indeed, right up to the point where the interval becomes infinity. This, of course, would be expected by the "interpretation" theory which has been presented here.

However, the present writer, in a previous study (Sylvester, 1960) seems largely to have overcome these difficulties by the following methods: (1) using four lights instead of two; (2) using *upward* runs only; and (3) instructing the subject to give a continuous running commentary on what he saw that is, it is the *experimenter* who determines between *phi* and *beta* movements. Recording the results of upward runs only would have the effect of displacing thresholds slightly, due to time error, but since it would displace both bottom and top thresholds in the same direction, it becomes irrelevant for the purposes of the present experiment.

When four lights, in square formation, are used, flashing on and off in succession round the square, *phi* movement is rarely seen and even when it is, it is *circular*. Straight-line optimal *beta*-movement occurs very suddenly and very clearly at the crucial time-interval when shortening the interval from succession (Sylvester, 1950). Very few subjects fail to report *beta* movement with the four-light set-up.

Method

Four circular light-boxes (made from 2 m diameter pill-boxes) were arranged at the corners of an upright square ABCD of side 6 in. The light-boxes were fitted with small 90 volt neon bulbs which could be flashed successively (A, B, C, D, A, etc.) by means of a rotating eccentric arm actuating four contacts equidistantly spaced. The front of each light-box was covered by a lid which had cut in it a circular aperture of either 1 mm or $^1/_2$ in. For part of the experiment the lids were removed to give an aperture of

2 in. The time-interval between lights (ti) was varied by adjusting the speed of the motor driving the arm and this motor was geared to a small direct-current permanent-magnet electric generator which activated, through a long cable, a calibrated galvanometer and so continuously indicated the time-interval between successive light-stimuli. The eccentricity of the cam was arranged so that the duration of each light was half of the time-interval between successive ones. The timing mechanism was situated in a room other than the experimental one to prevent the use of auditory cues and the experiment was carried out in the dark, the experimenters' light being adequately screened. The lights were presented flashing in rotation clockwise.

Subjects were seated so that their eyes were approximately 8 ft from the centre of the display and level with it. When the subject was seated, the experimenter said: "In a moment or so, I shall switch out the room light. Then you will see some light or lights in front of you and I want you to tell me what it is you see. Don't try to guess what you think you *ought* to see, but describe how the display appears to you. I am specially interested in any changes which may appear to take place, so please try to keep up a continuous running commentary — keep talking all the time." The experimenter then switched out the room light and switched on the apparatus. The time-interval between successive lights was slowly shortened, from infinity to 15 msec. The point where the subject first reported *beta* movement — as distinct from *phi* or anything else — was taken as his succession/movement threshold and the point where he reported seeing all four lights together was taken as his movement/simultaneity threshold (subjects did not, of course, know the expression "*beta* movement", their actual verbal reports were in terms of "Oh, now it's one light dodging in a square").

Eighteen subjects were used, 6 being tested with a light aperture of 2 in., 6 with a light aperture of $^1/_2$ in. and 6 with an aperture of 1 mm. Each subject was tested three times, once under Dexedrine (dexamphetamine sulphate, 10 mg), once under Sodium Amytal (195 mg) and once under a placebo; each group of 6 subjects being arranged in a balanced incomplete block so that every order of drug occurred once. Subjects used were all female student actresses, aged 19—25 years.

Results

As can be seen from Tables 4 and 5 and Fig. 5, the depressant drug Sodium Amytal, compared with Dexedrine and/or a placebo, lowered the simultaneity/movement threshold and raised the succession/movement threshold i.e. reduced the range of conditions over which apparent movement was seen.

An analysis of variance carried out on this range of time-interval showed drug-effect to be significant at the 0·01 level ($F = 10{\cdot}452$); the difference between subjects to be significant at the 0·05 level only ($F = 1{\cdot}941$) and drug-order and practice-effects to be not significant.

The difference of range between different drug conditions, however, is mostly due to the differences of the *bottom* threshold (this can clearly be seen in Fig. 5). There is no difference at the top threshold (i.e. movement-simultaneity) between Dexedrine and placebo and only a comparatively

TABLE 4

Lower thresholds for apparent movement in milliseconds

		Dexedrine	*Placebo*	*Amytal*
Lights 2 in. diameter (N = 6)	M	178	164·2	126·5
	6	36·0	34·3	15·95
$^1/_2$ in. diameter (N = 6)	M	147·7	151·2	147·3
	6	40·7	29·7	47·7
1 millimetre (N = 6)	M	171·8	145·0	133·3
	6	32·5	17·5	27·6
Grand Mean		165·8	153·5	135·7

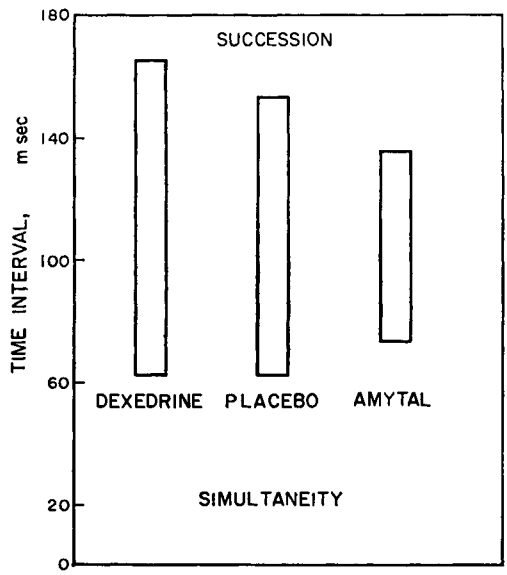
small difference between Amytal and the other two. This would seem to be good evidence that the drugs are having their effect on the central interpretative mechanism — the analyser or "computer" rather than on physiological cues or even the reception of these at the afferent endings —

TABLE 5

Upper thresholds for apparent movement in milliseconds

		Dexedrine	*Placebo*	*Amytal*
Lights 2 in. diameter (N = 6)	M	49·2	49·5	54·2
	6	11·7	11·06	10·7
$^1/_2$ in. diameter (N = 6)	M	69·5	73·8	87·3
	6	20·2	11·2	15·4
1 millimetre (N = 6)	M	69·0	65·2	78·3
	6	11·3	13·2	17·6
Grand Mean		62·6	62·8	73·3

using the model put forward in this study, it is the actual workings of the perceptual computer which is affected, more than the data fed into it or even its ability to receive data. It should be noted that the stimulant drug Dexedrine affected *only* the bottom threshold.



FG. 5. Histogram showing the range of time-interval over wich apparent movement was seen under different drug conditions.

It will be observed that the results for a light-aperture of $^1/_2$ in. do not fit between the 1 mm and 2 in. apertures, as might have been expected (see Fig. 6). Korte's laws would predict a variance with aperture-size, of course, but it is not easy to see why the $^1/_2$ in. size should be so different. Discussion of this in the present article is, if not irrelevant, at least fruitless in the absence of necessary data. What is needed here is much more experimentation.

It may be thought to be a weak point of hypothesis-construction where, at the beginning of this section, it was stated that mammals would "obviously have a drive" towards interpreting present sensory stimuli in terms of "real life", for no data were offered in support of this claim other than a rather general argument from "ordinary life" which might well be, in any case, interpreted as a matter of *habit-strength* rather than drive. There lies in the present experiment itself, however, a source of relevant data. Although in the 4-light set-up used seeing movement is continuous from the bottom threshold to the top one, the *kind* of movement varies. This will be elucidated in the next section, where it will be argued that the particular patterning of the changes in the kind of movement seen gives evidence in support of the hypothesis of a drive towards actuality. Further, whether or no stimulant–depressant drugs affect drive level in perception is very relevant

to the general argument of a central perceptual computer; the following section describes an experiment to show whether they do or not.

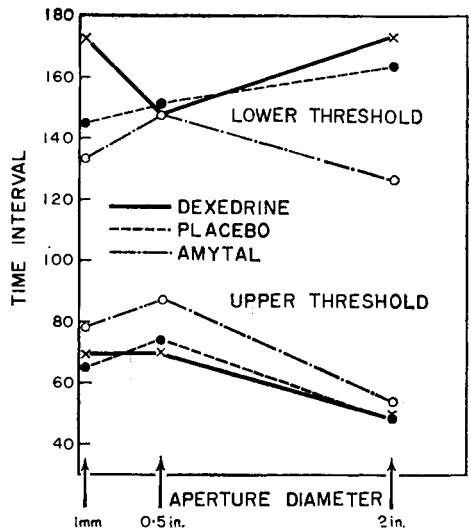


FIG. 6. Graph showing the effects of aperture-size on the upper and lower thresholds for apparent movement.

Variations of Type of Apparent Movement in the Four-Light Situation

If four lights are disposed at the corners of a square and flashed successively in rotation, then, as the time-interval between them is shortened from infinity, seeing the lights on and off in succession is followed by seeing one light tracing out a square; then there comes a sudden change to seeing *two* lights, either bouncing up and down the two vertical sides of the square or else oscillating to and fro along the diagonals and this continues until simultaneity is reached, (Sylvester 1960). Further, if the time interval is kept constant somewhere within the range over which the two-light *beta* movement is seen, then there is experienced a continual change-over from one patterning to another (from up-and-down vertical sides to to-and-fro horizontal sides, etc.), much in the manner of the alternations seen with the Necker Cube.

In the literature, the most common explanation for the Necker Cube phenomenon is either in terms of some kind of cortical-field theory or in terms of satiation theory — that the alternations are due to the building up of "resistance" or "inhibition". Supporters of the simple, physiological, theory of apparent movement would undoubtedly apply this explanation to the four-light alterations. There are, however, a number of facts about this four-light phenomenon which simply will not square with the satiation hypothesis.

In the first place, when two lights are seen moving, they are seen to move regularly and evenly to and fro or up and down. Now there is something

very odd about this, if we bear in mind Korte's laws which state that time-interval between lights and distance-apart of lights are critical parameters, and it needs careful consideration.

Let the square on which the lights are placed be called A B C D, and consider it when upright and when two lights are seen moving up and down the vertical sides, the lights actually being flashed successively in a clockwise direction. That the appearance of light C 60–100 msec after light B should produce apparent movement from B to C is only to be expected, but light B does not again appear until after an interval four times as long as the B—C interval. Why, therefore, should there be movement from C to B apparently at the same speed as, and quite regular with, the B to C movement? (The movement is strikingly clear and looks like a ball bouncing regularly up and down.) Further, it is light D which comes on after C, yet instead of movement from C to D, at intervals shorter than about 130 msec, what is seen is movement from *A* to D. The seen movement from C to B, A to D, etc. is evidently illusory on a way which cannot be accounted for on physiological grounds; it would seem to be a function of interpretation and hence primarily a matter of association-pathways and previous experience.

Secondly, if the lights are disposed at the corners of not a square but a rectangle, the up-down or to-and-fro movements and the alternations of the patterning still persist, regardless of the shape of the rectangle. Obviously this accords neither with any kind of simple physiological theory nor with Korte's laws and one must invoke some kind of central interpretative process, as in the constancies, to account for it.

Accordingly, what seems a more plausible theory to account for the alternation phenomenon may be stated as follows. The mere receiving of sensory impression is biologically useless; what is essential to adaptation is the giving of *meaning* to the kaleidoscope of received stimuli. Stimulus-patterns have to be fitted into a total field of comprehensibility. In ordinary life, this is what we do, continually and continuously, in perception. When we encounter a pattern that is not clear, we automatically seek further data which will settle doubt — we "strain our eyes", we move the head, try another angle, bring other senses into play. Whether this activity is regarded as a matter of *drive-level* or as *habit-strength* does not matter; it is what occurs. And one can be sure, then, that if an individual is presented with a stimulus pattern which is ambiguous, he will continually try "seeing it in a different way", using all the "different ways" that are possible, until either a solution is reached or fatigue sets in or frustration produces some kind of interfering activity. Since the four-light apparent movement situation is completely ambiguous, subjects alternate in the way they see it in an endeavour to give it reality.

If this account is true, one can put forward the hypothesis that *depressant drugs (which reduce both drive and habit-strength) will reduce the rate and variety of alternations.* Stimulant drugs presumably would increase them. This is the hypothesis which was tested in the experiment to be described.

It should be noted that the satiation theory gives an exactly opposite prediction and, therefore, this experiment is a crucial one for determining between the two theories.

Method

With the apparatus described in the previous section, the time-interval was set at 120 msec and subjects were required to gaze at the display for a period of 2 min, giving a continuous running description of what they experienced. The number of alternations reported in this period was recorded. Twelve subjects were used, each receiving, on different days, 10 mg. of Dexedrine, 227 mg of Sodium Amytal or a placebo. Light-apertures of 2 in. and 1 mm were employed.

TABLE 6

Mean number of alternations in 2 mm period under different drug conditions
(N = 12)

Aperture size	*2 in.*	*1 mm*	*Grand Mean*
Dexedrine	25·6	21·3	23·5
Placebo	19·0	17·3	18·2
Amytal	17·4	14·7	16·0

Results

As will be seen from Table 6 and Fig. 7, Dexedrine had the effect of considerably *increasing* the number of alternations seen, while the effect of the depressant drug was to reduce the number, compared to the number

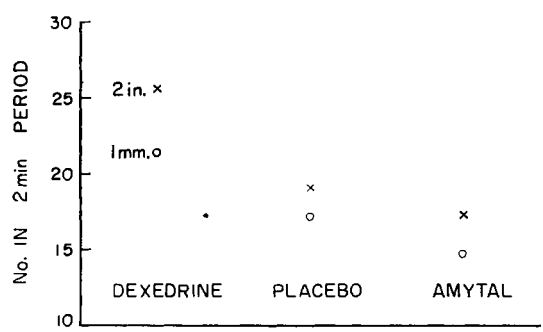


FIG. 7. Graph showing the effects of the drugs on the number of alternations seen.

experienced with the placebo condition. The effects are the same for both aperture-sizes. The results, that is, confirm the hypothesis drawn from drive-level/habit-strength and the model of the perceptual computor, and run counter to the neural-satiation theory.

However, this is but one experiment – and with a phenomenon not previously investigated except by the present writer. Clearly it would be as well to try and relate the phenomenon in question to other, known variables. Also, this general discussion of inhibition would be incomplete without carrying it into the field of individual differences, in no matter how limited a way. Accordingly, in the next sections an attempt will be made to follow Eysenck a little way into connecting personality with perception.

The Alternation Phenomenon, Extraversion and Rigidity

"Spatial inhibition effects in the perceptual field are stronger in the case of extraverts ... than in the case of introverts ..." runs one of Eysenck's postulates and another is to the effect that depressant drugs produce cortical inhibition. That is, depressant drugs act like extraversion and if it has been found that depressant drugs produce a certain effect, one should, accordingly, expect this effect to be correlated with extraversion. We have already found, in the previous section, that a depressant drug *lowers* the rate of alternation in the four-light situation: is there, then, a negative correlation between rate of alternation and amount of extraversion?

A personality variable which might well be equally important is *rigidity*. Does rigidity also have a negative correlation with rate of alternation?

An attempt to answer these questions was made with the following experiment.

Method

The apparatus was as used in the earlier experiments here described — four lights disposed about a 6 in. square. The time-interval between successive light-flashes was kept constant at 100 msec for 2 min for each subject. Subjects were 32 male engineering apprentices aged 17–25. As before, each subject was required to give verbally a continuous description of what he could see and changes were recorded. All subjects were given the Maudsley Personality Inventory, a test of introversion–extraversion and also of neuroticism. In addition, each subject completed two paper-and-pencil rigidity tests, the C.P.I.R. (Eysenck, 1952) and the N.R. *(ibid)*. Finally, for good measure, they were tested on the Archimedes spiral. Eysenck has said that the duration of persistence of the after-effect of a rotating spiral should correlate negatively with extraversion — "much inhibition and short duration of after-image would be expected in the hysterico-psychopathic, the extraverted, and the brain-damaged group." (Eysenck 1957, p.164). Stimulation-time for the spiral was one min. Product–moment correlations were calculated between these six measures.

TABLE 7

Means of subjects' extraversion, neuroticism and rigidity Scores, with rate of alternation

$N = 32$	*Mean*	*Stnd. Devtn.*
M.P.I. Extraversion	31·9	9·5
M.P.I. Neuroticism	19·8	6·2
N.P. Rigidity	14·1	5·6
C.P.I.R. Rigidity	15·8	4·9
Spiral (time in secs.)	15·6	6·3
Alternations	13·5	8·3

TABLE 8

Intercorrelations of test scores, spiral persistence-time and rate of alternation

	Alternations	*Extraversion*	*Neuroticism*	*C.P.I.R.*	*N.R.*	*Spiral after-effect*
Alternations		0·4380	−0·1906	−0·0614	−0·1776	−0·1371
Extraversion (M.P.I.)			−0·0778	−1·638	−0·3036	0·0311
Neuroticism (M.P.I.)				0·1872	−0·1112	0·0469
Rigidity (C.P.I.R.)					0·3586	0·0683
Rigidity (N.R.)						0·3721
Spiral after-effect						

Results

Table 7 gives the means of the subjects' scores on the tests, their mean spiral after-effect persistence time and the mean rate of alternation in the four-light apparent movement set-up. Table 8 gives the correlation matrix

of the sets of data obtained. The correlation between the number of alternations and extraversion, as measured by the M.P.I. was significant (0·05) and *positive*. Apart from the expected one between the two measures of rigidity, this correlation is the only significant one. Predictions from Eysenck's personality postulates *plus* the assumption that satiation is responsible for the alternation phenomenon are borne out in this experiment, but it will be remembered that in our drug experiment, the findings went counter to a similar combination of Eysenck's drug postulate and the satiation hypothesis. The assumption that satiation is, in fact, responsible for the alternation phenomenon was, of course, questioned by Eysenck, Holland and Trouton (1957) who found no evidence for drug effects either way in their own experiments. It is impossible to know whether their results and those here reported are really in conflict, as the present method of measuring reversible perspective phenomena is different from theirs, and may involve quite different mechanisms.

What predictions, then, in the field of individual differences would be made from the position taken in this paper – the model of a "perceptual computor"? It is a fundamental plank in this concept that the "computor" is "programmed" by previous experience – that perception is largely *learned*. It follows that where two individuals differ on some factor in perception, they must have had some related specific difference in their previous individual experiences, and also, of course, the same factor of experience will probably affect some aspect of the individual's present motor behaviour.

To try to sort out the mass of units of previous experience, of course, is an enormously difficult task, perhaps impossible. However, it was decided to make some attempt, for there seems no reason why some kind of start to the problem, no matter how small, should not be made. Accordingly, the following hypotheses were formulated.

(1) If the perception of apparent movement is a function of previous perception of real movement, then one would expect that those individuals who have had more experience of real movement, and responding to it, would see apparent movement more readily than those who have had less. It is postulated, that is, that individuals seeing apparent movement most readily would be found to have had a childhood most involved with ball-games, mechanical toys and activities of an extraverted nature.

(2) Perception cannot be isolated from response. A creature with a central nervous system is a mass of feed-back circuits, a mass of servo-mechanisms. The tracking-task type of experiment well illustrates this for here the subject simply *is* a servo-machine. Servo-mechanisms, of course, – at any rate those concerned with tracking objects moving according to some regular function or functions (e.g. the ones used with anti-aircraft guns) – essentially include a computer, on the speed and accuracy of which depends the efficacy of the servo-mechanism effecter. If, then, we have a tracking-task with a regular and hence readily learnable track, one may predict that variations of performance between individuals will be associated with variations in efficiency of the central computer or perceptual analyser. Since we have already claimed that such variations are a function of previous

experience, we shall naturally expect the same kind of relationship between this and performance as outlined in (1). The so-called pursuit-rotor (to be described) provides an experiment by which these predictions can be put to the test. It will be noted that no relationship would be expected on any first trial on this apparatus since before the experimental situation which we have in mind can be reached, the movement-function of the track has to be learned. On later trials, however, it is predicted that there will be a positive correlation between performance and other perceptual tasks, such as the readiness to observe apparent movement and the drive-level/habit-strength of attempts to fit ambiguous stimulus patterns to reality. It is to be noted that if individuals do differ in the permanant efficacy and programming of their perceptual analysers, then one would expect the effect to show itself over a wide variety of perceptual-motor skills: to be reflected, for example, in performance at ball-games and similar activities.

Method:

The following measures were obtained:

(1) Bottom and top thresholds of apparent-movement in the four-light situation.

(2) Rate of alternation of 2-light patterning in the four-light situation.

(3) Number of errors on first and last runs on the pursuit-rotor tracking task.

(4) A score derived from a questionnaire concerned with the amount of perceptual-motor activity in childhood and with early ability at ball-games.

The apparatus used for the apparent-movement measures was that previously described in this study. The time interval was slowly shortened from infinity to past the point of simultaneity, then lowered again and set at 100 msec for a period of 2 min. As before, subjects gave continuous verbal reports.

The pursuit-rotor used was somewhat similar to a gramophone turntable — a rotating disk of insulating material with a $^1/_2$ in. diameter circular metal plate set in it towards the circumference. The turntable is set to revolve at a speed of one revolution per sec. The subject, who stood in front of and over the turntable was provided with an articulated rod with a metal tip which was wired into the recording mechanism. He was required to maintain contact between the stylus and the metal plate on the turntable and the number of times he failed to do so was recorded electrically on a tachometer. (This procedure differs somewhat from that described by Eysenck (1957), where what was recorded was *the total time on target* per successive 10 sec periods).

All subjects were given a score derived from the following question schedule, in the manner indicated. Subjects were 30 engineering apprentices.

The Question Schedule

(1) How many siblings?	Score 1 each
(2) Where did you live as a child?	Country scores 1, Town 0
(3) Did you read much as a child?	Yes = 0, No = 1
(4) Did you "play out" much?	Yes = 1, No = 0
(5) How much time spent playing out?	Most 2, No 1, Little 0
(6) What was favourite toy when very young?	Mechanical 1–3, Soft 0
(7) Father's occupation?	0–3
(8) What kind of play-activity before 11 years?	0—3
(9) How good at games at school?	0—2
(10) What games best at in school?	Football 1, cricket 2
(11) How good at handcrafts at school?	0–2
(12) What games played now?	Cricket, t-tennis etc. 2, Others 1
(13) How well do you play table tennis?	Poor 0, Av. 1, Good 2
(14) How well lawn tennis?	Not 0, Some 1, Fair 2
(15) Are you good with hands or poor?	0, 1, 2
(16) Do you like making things?	Yes 1
(17) Do you like repairing things?	Yes 1
(18) Have you ever been described as clumsy?	No 1
(19) What would you like to be? a magic wish!	0–2
(20) What kind of handcrafts now?	1 each

Results

Only the first 8 questions of the questionnaire refer to the subject's environment before he was about 11 years old; therefore, the total score of these was used separately in the correlation calculations, as well as the total score of all 20 questions. The rate of alternation correlated significantly with both the 8-question and the 20-question scores, the latter yielding the remarkably high figure of 0·81. The correlation matrix is given in Table 9. As will be seen from this, the alternations also correlate significantly with performance-skill on the pursuit rotor, the *last* run, as was predicted from the *learning* theory of perception. The correlation between alternations and performance on the first run on the pursuit-rotor was positive but not significant. From the general theory put forward in this study, one might have expected a negative correlation between rate of alternation and the lower threshold, but this was not found. There was, however, a significant negative correlation between the lower threshold and the last-run pursuit-rotor task, which fits in well with the hypothesis put forward. The top threshold yielded no significant correlations. The extraversion–introversion scale of the M.P.I. gave no correlation over 0·2, well below significance.

TABLE 9

Intercorrelations between test-scores, tracking-task, experience-questionnaire, thresholds and alternations.

	B	*C*	*D*	*E*	*F*	*G*	*H*
A Bottom threshold	0·3312	0·0893	−0·0173	*−0·3820*	0·0019	−0·2143	0·1070
B Top Threshold		0·0175	−0·0534	−0·1154	−0·0734	−0·0156	0·0807
C Alternations			0·3124	*0·4088*	*0·8109*	*0·5507*	−0·1908
D Tracking task lst.				*0·5860*	0·3299	−0·1592	0·0626
E Tracking task 2nd.					*0·4637*	0·2143	0·1637
F Experience Questionaire (20 questinons)						0·2070	−0·1084
G Experience Questionaire (8 questionns)							0·2070
H M.P.I. Extraversian scale							

Figures in italics are statistically significant.

(Note. Since the scores for the tracking task were obtained by counting the number of errors, the signs for D and E above have been reversed so as to read as SKILL in performance.)

CONCLUSIONS

The results of the six experiments described in this study may be briefly summarized as follows:

(1) Size-constancy, in conditions of cue-reduction, is diminished by Sodium Amytal, a depressant drug. The drug may produce the effect either by reducing the physiological cues arriving at the cortex or by reducing the efficiency of the cortical analyser – the processes of calculation of, or compensation for, distance.

(2) Shape constancy is equally reduced by the depressant drug. The suggestion here is that it is the previously learned associations between retinal-shape, tilt and real shape which are being interfered with. Probabilities seem to be on the side of a reduction of efficiency of the "perceptual computer" rather than on reduction of physiological cues, but the experiment is not conclusive.

(3) The range of time-interval between lights over which apparent movement is seen is reduced by Sodium Amytal. The reduction is accounted for more by a raising of the bottom threshold (succession to movement) than by the lowering of the top threshold (movement to simultaneity), although this also occurs. It was argued that apparent movement at the bottom threshold is a phenomenon much more akin to the constancies than to "figural after-effects" under which title it has been more commonly included, particularly by *Gestalt* psychologists. The raising of the bottom threshold cannot be accounted for peripherally since the time intervals are too long, nor can it be a matter of reduction of physiological-cue (in the experimental set-up used no cues are available except those covered by Korte's laws and these did not hold in the particular situation used).

There remains an explanation in terms of a reduction of the efficiency of the "perceptual computer." It is argued that whether this is described as a reduction of "drive-level" or as a reduction of "habit-strength" or as an interference with the "programming" of the "computer" is irrelevant since there is no meaning to be attached to distinctions between these phrases.

(4) A phenomenon is described where, in a four-light apparent-movement set-up, there occur alternations in the patterning of the precise type of movement seen similar to those experienced with the Necker Cube. "Satiation theory" would predict an increase in the rate of these alternations with a depressant drug; the "learning theory" urged in this discourse predicts a decrease. In fact, the depressant drug *lowered* the rate of alternation while the stimulant drug Dexedrine increased it.

(5) The rate of alternations referred to above was correlated with upper and lower thresholds for apparent movement, with performance on a tracking task and with a score derived from a question-schedule concerned with previous experience in sensory–motor activity. A high, significant and positive correlation was found between rate of alternation and early environment concerned with previous experience of sensory–motor activity. The rate of alternation also correlated positively with performance on the pursuit-rotor after a period of initial practice, a result which supports the view that the competancy of the perceptual half of sensory – motor function depends upon the efficiency of the perceptual "computor", and that the competency of this depends very largely on how it has been "programmed", i.e. depends on learning.

(6) A new theory of perception, paticulary relevant to apparent movement and the constancies, has been put forward. This has been supported by the data.

Chapter 12

BRAIN DAMAGE AND DEPRESSANT DRUGS: AN EXPERIMENTAL STUDY OF INTERACTION

MARYSE CHOPPY*

and

H. J. EYSENCK

Introduction

Throughout that part of the literature on depressant drugs which is concerned with personality differences there runs a vague belief that possibly the effects of these drugs (particularly alcohol) do not only push introverted, ambiverted and extraverted subjects in the direction of greater extraversion, but also that the strength of this "push" is in some way related to the position of the subject on the extravert–introvert dimension. This belief usually takes the form of expecting extraverts to show *stronger* drug effects, i.e. they are not only nearer the sedation threshold to begin with, but they also approach it more quickly than introverts after taking an equivalent dose of drugs. No proper test of this hunch (it can hardly be called a hypothesis) appears to have been carried out, and the present study was arranged in an attempt to provide some evidence on this, as well as on some other points.

The groups actually chosen for the experiment were not, however, made up of extraverted and introverted subjects. As pointed out in the first chapter, the particular point of the excitation–inhibition balance represented by extraverts can equally well be represented by other groups, such as brain-damaged subjects, and in this chapter we are presenting results obtained by comparing groups of brain-damaged and non-brain-damaged subjects.

As our main interest will be in the acquisition and extinction of conditioned responses, it may be interesting to the reader to compare some data from brain-damaged and non-brain-damaged subjects obtained by means of the eyeblink conditioning apparatus, with the data on extraverted and introverted subjects, obtained by means of the same apparatus and procedures, given in the first chapter of this book (Fig. 3). We would expect, by and large, that the brain-damaged subjects would give records similar to those of the extraverts; we would expect the non-brain-damaged

* This experiment was carried out in the Service de Neurochirurgie at the Salpétrière Hospitál in Paris, and we are indebted to Dr. G. Le Beau for his permission and help. We also wish to thank Mme. N. Zimbacea for her help in testing the potients.

subjects to give records half way between the normal extraverted and introverted groups, the reason being, of course, that such a non-brain-damaged group would, on a random sampling basis, contain no more extraverted than introverted subjects. Fig. 1 shows data reported by Franks (1959); the subjects were 17 brain-damaged and 52 non-brain-damaged mental defectives (mental defect as such does not appear to affect conditioning). It will be seen that our prediction is fully verified; "the correlation coefficient between the acquisition scores and organic damage.....was 0·357, which is significant at the 0·01 level, and the correlation between extinction scores and organic damage.....was 0·274, which is significant at the 0·05 level. Hence it is quite clear that patients with a diagnosis of organic damage fail to acquire stable conditioned responses as well as patients without this diagnosis."

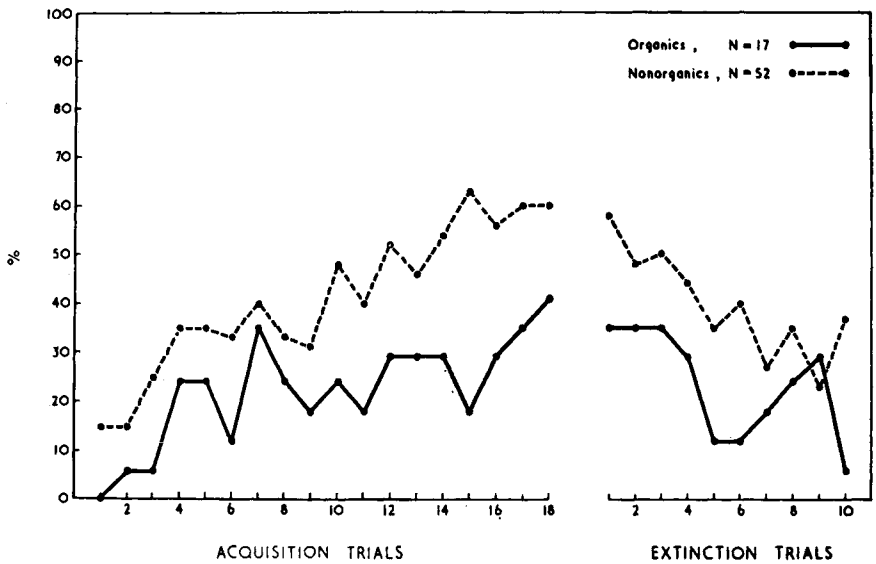


FIG. 12.1 1. Acquisition and extinction of conditioned eyeblink responses by brain-damaged and non-brain-damaged subjects. From Franks, 1959.

This study is, of course, not the only one to demonstrate the relationship between extraversion and brain damage, or even to demonstrate the fact that brain damage disturbs the acquisition and retention of conditioned responses; a survey of much of the literature has been given in Eysenck (1960). We are, therefore, on relatively safe ground in using groups of brain-damaged subjects to exemplify that part of the excitation–inhibition continuum which is characterized by high inhibition and low excitation.

There is a good deal of evidence also linking depressant drugs with a *lower rate* of conditioning and stimulant drugs with a *higher rate;* some

of this is referred to in papers by Franks and Laverty (1955), and Laverty and Franks (1956). Franks and Trouton have performed a direct test of the hypothesis that conditioning is affected by these drugs in a predictable manner, using the identical eyeblink conditioning apparatus used in the brain damage experiment shown in Fig. 1. The results of this study are shown in Fig. 2. (Franks and Trouton, 1958), and it will be seen that normal groups of subjects, on the average neither extraverted nor introverted, can be made to give conditioned responses resembling those of typically extraverted or introverted groups by the administration of Dexedrine or Sodium Amytal.

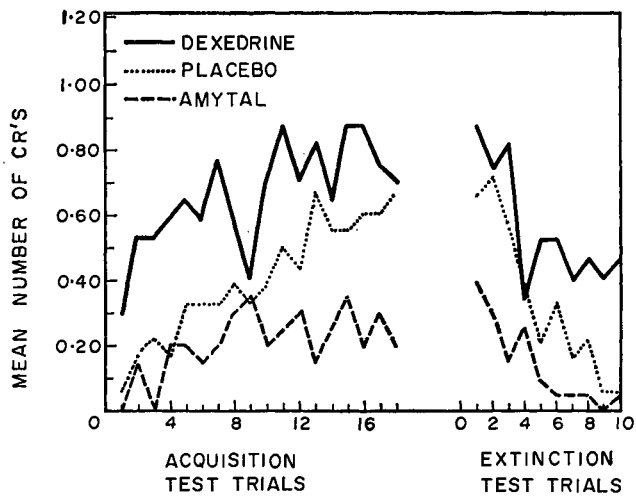


FIG. 2. Acquisition and extinction of conditioned eyeblink responses under placebo conditions and after drug administration. From Franks and Trouton, 1958.

The Experiment

The design of the experiment uses three brain-damaged and three non-brain-damaged groups, henceforth to be called the "organic" and "non-organic" groups. One organic and one non-organic group constituted the control groups; they were given placebos instead of the active drugs administered to the other groups. One organic and one non-organic group were administered meprobamate (Equanil Byla). The subjects received three doses of 400 mg of meprobamate each day for 4 days. On the day of testing, they received these three doses in the morning, the test itself beginning early in the afternoon. One organic and one non-organic group were administered amylobarbitone sodium (Eunoctal Roussel). Treatment consisted of 100 mg of Sodium Amytal administered each evening for 3 days. On the day of testing, the patients received the same dose in the morning and again at noon, the test beginning 2 hours after the administration of the second dose; they thus received approximately three grains on the day of testing.

Each group consisted of 20 subjects, making a total of 120 subjects in all. The organic groups consisted of patients suffering from generalized brain damage rather than from localized lesions; all operations were external to the substance of the cerebral hemisphere. Some of the patients were suffering from fractures of the skull, or from extra-dural haematoma. The control group was made up of patients undergoing spinal operations in the lumbro-sacral region (laminectomies) for various disk lesions. They were tested on the ninth or tenth day after the operation.

The following tests were administered to the 120 subjects of this experiment.

(1) *The Maudsley Personality Inventory* (Eysenck, 1959) – This inventory, which consists of 48 questions, gives a reasonably reliable and valid measure of neuroticism and extraversion. It was translated into French for the purpose of this experiment and the French translation is included as an appendix to this chapter.

(2) *The Epstein test of over-inclusive concept formation* – This test was developed by Epstein (1953) as a direct measure of "over-inclusion" in concept formation as defined by Cameron (1947). This test consists of a list of 50 words. Following each stimulus word there are 6 response words, including the word "none." The task of the subject is to underline all those response words which are an essential part of the concept denoted by the stimulus word. In view of the greater "over-inclusiveness" of schizophrenics, Epstein predicted that patients would underline more response words than normals. He found this to be true, and Payne and Hirst (1957) found even higher "over-inclusion" scores in a group of endogenous depressives. Later studies by Payne, Matussek and George (1957), and Payne and Hewlett (1960) give rather contradictory results, the former showing very high, the latter rather low "over-inclusion" scores for schizophrenics. The test can also be scored for "under-inclusion", i.e. a tendency for the subject to underline too few of the appropriate responses. This test was translated into French, and the translation may be obtained from Dr. Choppy.

(3) "*Reaction time experiment*" – Sixty-four auditory stimuli were given at irregular intervals, identical for each subject; reaction tim es were measured with an accuracy of approximately 1/100 sec. Each subject was demonstrated the test and gave 10 to 15 reactions to make sure that the procedure was understood. Subjects were instructed to press down a morse key the moment they heard the stimulus; they had a finger on the key all the time, and no warning signals were given.

(4) *G.S.R. conditioning* – This experiment was carried out along the usual lines. The subject was connected to the psychogalvanometer and allowed to rest quietly for a while until he reached a suitable "resting level". Several auditory stimuli were then delivered over earphones until no further responses to these were observed on the ink writer which recorded variations in the subject's resistance. After this, the acquisition period of the experiment began in which an electric shock of moderate severity followed the tone after 5 sec. The tone–shock sequence was repeated at irregular intervals which were, of course, identical for each

subject; ten such repetitions constituted the *acquisition* phase of the experiment. During the *extinction* phase ten tones were given at irregular intervals, but this time not followed by reinforcement (shock).

For the purpose of scoring we noted the number of conditioned responses for each subject, that is to say, the number of responses in the acquisition phase of the experiment which occurred between the tone and the shock; in the extinction phase we counted responses made to the tone within a period of 5 sec. Each response was then weighted according to a scheme which gives a greater weight to those *acquisition* responses which occur *early* in the series, and to those *extinction* responses which occur *late* in the series. Results have also been investigated without the use of this weighting procedure; they do not in any important way deviate from those obtained in the manner outlined above.

In addition to the four experimental tests subjects were administered the Wechsler Adult Scale as we wanted the groups to be reasonably matched on this variable, and not to include subjects of too low intelligence to understand properly the procedures of the experiment. We must now turn to a discussion of the results of the experiment.

Results

(1) *Maudsley Personality Inventory* — The results of both the extraversion and the neuroticism test are given in Table 1. An analysis of variance was performed on each of these two sets of scores, with the result that difference in extraversion between the groups was found to be significant at the 0·05 level and difference in neuroticism at the 0·01 level. It would appear that the organics in our experiment were slightly less extraverted and much more neurotic than the non-organics. The extraversion differences are separately significant by *t*-test only for the placebo group; there is a slight tendency for organics to become more extraverted and for non-organics to become less extraverted under the drug. These effects, however, are not significant, as shown by the interaction term of the analysis. On neuroticism too, the main gap between the organics and the non-organics is in the placebo group; the gap becomes distinctly smaller under the two depressant drugs. This is illustrated by the fact that the difference is independently significant on a *t*-test at the 0·01 level under placebo conditions, at the 0·05 level under meprobamate, and not significant under Sodium Amytal. However, here again the interaction term in the analysis of variance is not significant.

TABLE 1

M.P.I. scores: extraversion

	Placebo	*Meprobamate*	*Sodium Amytal*
Organic	22·85 ± 5·08	25·05 ± 6·02	24·95 ± 7·72
Non-organic	29·85 ± 11·06	26·95 ± 8·22	26·35 ± 8·56

M.P.I. scores: neuroticism

	Placebo	*Meprobamate*	*Sodium Amytal*
Organic	24·95 ± 14·95	25·95 ± 13·56	22·85 ± 13·28
Non-organic	12·40 ± 11·28	17·70 ± 12·22	15·25 ± 11·47

(2) *Epstein test* – The results of the Epstein test, both with respect to over-inclusion and under-inclusion, are set out in Table 2. It will be seen that there are no differences in the under-inclusion part, and indeed this score was only included for the sake of completeness; there was no rationale for expecting any difference to appear. As regards over-inclusion, however, there is a clear drug effect; both organics and non-organics have much higher scores under both meprobamate and Sodium Amytal than they do under placebo conditions. Both groups give almost identical scores under placebo conditions, and the drug effect will be seen to be much more marked for the organics than for the non-organics (the change in the direction of over-inclusion for the organics is 14·10 and 17·00 under the two drugs, while for the non-organics, it is 9·05 and 3·00, i.e. less than half). An analysis of variance discloses that a drug effect is fully significant at the 0·05 level, but that the interaction effect, although suggestive, falls short of significance.

TABLE 2.

Epstein test

	Over-inclusion		
	Placebo	*Meprobamate*	*Sodium Amytal*
Organics	29·45 ± 17·34	43·55 ± 22·32	46·45 ± 22·71
Non-organics	29·15 ± 15·49	38·20 ± 24·46	32·15 ± 12·12
	Under-inclusion		
	Placebo	*Meprobamate*	*Sodium Amytal*
Organics	21·50 ± 6.66	20·85 ± 6·25	22·85 ± 7·48
Non-organics	19·85 ± 6·74	20·70 ± 8·00	17·85 ± 4·74

(3) *Reaction Times* – The means as well as the variability *(sigma)* of the reaction time scores are given in Table 3. It will be seen that while

the non-organics are, on the average, very slightly faster, and while the drugs apparently produce a slight slowing down, these effects are much too weak to be taken seriously, and indeed an analysis of variance discloses no significant difference anywhere. This position is different, however, when we turn to *variability*, where an analysis of variance discloses that the organics give more variable reactions than do the non-organics.

TABLE 3

Reaction times: means

	Placebo	*Meprobamate*	*Sodium Amytal*
Organics	19·36 ± 4·94	20·10 ± 5·35	18·70 ± 4·17
Non-organics	15·41 ± 2·50	19·72 ± 7·34	19·94 ± 4·90

Reaction times: variability (σ)

	Placebo	*Meprobamate*	*Sodium Amytal*
Organics	6·30 ± 1·81	6·67 ± 2·86	5·61 ± 2·43
Non-organics	4·98 ± 1·35	5·16 ± 1·90	5·70 ± 1·80

(4) *Conditioning* — Table 4 gives the mean scores for the acquisition and the extinction phase of the experiment respectively. It will be seen that while the organics and non-organics of the placebo group do not differ to any extent on either acquisition or extinction scores, the *non-organics have much higher scores under drugs than do the organics.* This is shown very clearly in Fig. 3, which also shows that the drugs had relatively little effect on the non-organics group. An analysis of variance was done for both the acquisition and extinction data. As far as the acquisition scores are concerned the differences between groups, the differences between treatments, and the interaction were all significant at t levels intermediate between 0·10 and 0·05. As regards the extinction phase the difference between groups was significant at well above the 0·01 level and the interaction term just fell short of significance at the 0·05 level; treatment effects were significant at the 0·10 level.

It will be clear from Table 4 that the original scores are rather skewed, and also that the means and variances are highly correlated. A square-root transformation of the raw data was, therefore, carried out, and the transformed scores reanalysed. This increased the significance of the results beyond the 0·05 level for groups and interactions alike, thus leaving

little doubt that in this experiment (a) organics acquired conditioned responses less quickly and (b) that both with respect to acquisition and extinction this lower conditionability is depressed proportionately more in organics than in non-organics. The data thus confirm our major hypothesis (or hunch) with respect to the interaction between drug effect and brain damage.

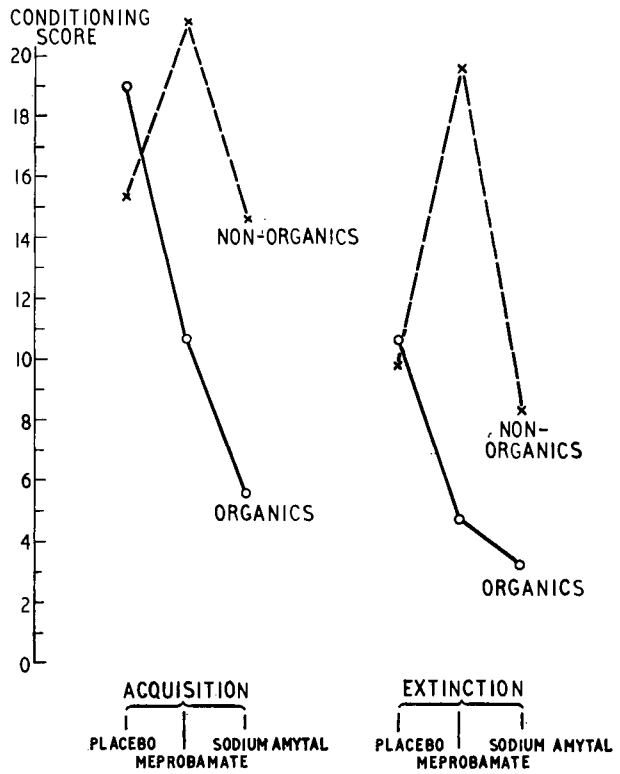


FIG. 3. Acquisition and extinction scores (G.S.R.) of brain-damaged and non-braine damaged subjects under placebo conditions and after drug administration.

TABLE 4

Conditioning: acquisition

	Placebo	*Meprobamate*	*Sodium Amytal*
Organics	19·00 ± 15·98	10·70 ± 13·89	5·55 ± 10·83
Non-organics	15·40 ± 15·70	21·05 ± 16·42	14·60 ± 16·61

Conditioning: extinction

	Placebo	*Meprobamate*	*Sodium Amytal*
Organics	10·70 ± 13·17	4·90 ± 5·02	3·35 ± 7·88
Non-organics	9·95 ± 13·64	19·60 ± 18·06	8·45 ± 15·71

Discussion

In evaluating the data here presented, it should be borne in mind that the type of brain-damaged patient studied here is rather different from the type of brain-damaged patient on whom the original work linking brain damage and extraversion was done. Most of the original studies were concerned with damage to the prefrontal lobes, either through accident or some form of lobectomy operation; Willett, (1960), LeBeau (1956), and Petrie (1952), among others, have attempted to relate directly such behavioural tendencies as extraversion–introversion and neuroticism to specified areas in the cortex. These areas were not, on the whole, touched in the patients making up our present groups and it will be noticed that on the questionnaire, at least, the organics come out less extraverted than the controls. Equally it will be seen that as far as the placebo group is concerned the organics were not inferior to the controls with respect to conditioning; it required the administration of one of the two depressant drugs used in this experiment to bring out the gross inferiority of the organics as far as both acquisition and extinction of conditioned responses are concerned.

The experiment thus leaves unanswered certain questions which only further work can resolve. It suggests that not all parts of the brain are strongly related to the dimension of extraversion–introversion, and indeed any such doctrine of equi-potentiality would, of course, be quite unlikely to be true in any case (Eysenck, 1957). Nevertheless, even such quite non-specific brain damage as studied here apparently has potentiating effects for strengthening the action of depressant drugs (and possibly other drugs as well), when under placebo conditions no effects are observable.

MAUDSLEY PERSONALITY INVENTORY

(version française)

NOM:	PRENOM:	AGE:
Profession:	Sexe:	Date du test:

CONSIGNES: Voici plusieurs questions concernant votre facon de penser et d'agir. Apres chaque question il y a un "Oui", un "?" et un "Non".

Tracer un cercle autour du "Oui" ou du "Non" suivant que la question correspond ou non à votre facon habituelle d'agir ou de sentir. Si vraiment vous n'arrivez pas à vous décider, tracez un cercle autour du "?". Travaillez rapidement et ne passez pas trop de temps sur chaque question, c'est votre premiere impression qui nous intéresse et non le résultat d'une longue discussion intérieure.
Tout le questionnaire ne doit pas prendre plus de quelques minutes. Il n'y a pas de bonnes ou mauvaises réponses, ce n'est pas un test d'intelligence ou d'aptitude mais seulement une étude de vos habitudes.

Repondez à chaque question.

(1) Etes-vous très heureux quand vous êtès implique dans des projets qui réclament une action rapide? ..... Oui ? Non
(2) Est-ce que vous vous sentez quelquefois heureux ou quelquefois désespéré sans raisons apparentes? ..... Oui ? Non
(3) Est-ce que votre esprit vagabonde souvent quand vous essayez de vous concentrér? ..... Oui ? Non
(4) Est-ce vous qui prenez d'habitude l'initiative quand vous vous faites de nouveaux amis? Oui ? Non
(5) Avez-vous tendance à agir rapidement et sûrement? Oui ? Non
(6) Etes-vous souvent perdu dans vos pensées, même quand vous êtès supposé prendre part à la conversation? Oui ? Non
(7) Etes-vous parfois bouillonnant d'énergie et parfois au contraire très indolent? Oui ? Non
(8) Est-ce que vous vous considérez vous-même comme quelqu'un plein de vitalité? Oui ? Non
(9) Seriez-vous très malheureux si vous étiez empêché d'avoir de nombreux contacts sociaux? Oui ? Non
(10) Avez-vous tendance à être triste? Oui ? Non
(11) Avez-vous souvent des hauts et des bas dans votre humeur, motivés ou non? Oui ? Non
(12) Préférez-vous agir à faire des projets d'action? Oui ? Non
(13) Est-ce que vos rêveries ont souvent pour sujet des choses qui ne seront jamais réalisables? Oui ? Non
(14) Avez-vous tendance en société à rester en arrière-plan? Oui ? Non
(15) Etes-vous enclin à ruminer votre passé? Oui ? Non
(16) Vous est-il difficile de vous laisser aller complètement même dans une réunion très gaie? Oui ? Non
(17) Vous arrive-t-il de vous sentir "misérable" sans raison valable? Oui ? Non
(18) Avez-vous tendance à être consciencieux à l'excès? Oui ? Non
(19) Est-ce que vous pensez souvent que vous vous êtes décidé trop tard? Oui ? Non
(20) Aimez-vous les contacts sociaux? Oui ? Non
(21) Est-ce que souvent vos soucis vous empèchent de dormir? Oui ? Non
(22) Avez-vous tendance à limiter vos relations à quelques personnes de choix? Oui ? Non

(23) Est-ce que vous avez souvent un sentiment de culpabilité? Oui ? Non
(24) Prenez-vous toujours votre travail comme si c'était une question de vie et de mort? Oui ? Non
(25) Est-ce que vos sentiments sont assez facilement choqués? Oui ? Non
(26) Aimez-vous avoir beaucoup d'obligations mondaines? Oui ? Non
(27) Vous considérez-vous comme un individu tendu au sous-pression? Oui ? Non
(28) Dans une activité de groupe préférez-vous généralement en prendre la direction? Oui ? Non
(29) Avez-vous souvent ressenti des moments (périodes) où vous avez l'impression de solitude? Oui ? Non
(30) Avez-vous tendance a être timide en face d'une personne de l'autre sexe? Oui ? Non
(31) Est-ce que vous aimez vous abandonner à la rêverie? Oui ? Non
(32) Avez-vous presque toujours une réponse prête pour les remarques dirigées contre vous? Oui ? Non
(33) Passez-vous beaucoup de temps à penser au bon temps que vous aviez autrefois? Oui ? Non
(34) Est-ce que vous vous considérez comme quelqu'un qui prend les choses du bon côté? Oui ? Non
(35) Est-ce que souvent vous vous sentez fatigué et sans courage, sans raisons valables? Oui ? Non
(36) Avez-vous tendance à vous effacer méme quand vous êtes en société? Oui ? Non
(37) Après avoir passé un moment difficile, pensez-vous généralement â quelque chose que vous auriez dû faire mais dont la réalisation n'a pas été possible? Oui ? Non
(38) Pouvez-vous généralement vous détendre, vous laissiet aller et avoir du bon temps en joyeuse compagnie? Oui ? Non
(39) Est-ce qu'il vous arrive d'avoir des idées qui vous trottent dans la tête au point que vous ne puissiez dormir? Oui ? Non
(40) Est-ce que vous aimez le travail qui réclame une grande minutie? Oui ? Non
(41) Est-ce que vous avez déjà été préoccupé par une pensée sans intérêt qui persiste à revenir dans votre esprit? Oui ? Non
(42) Est-ce que vous avez tendance à prendre votre travail comme une routine? Oui ? Non
(43) Y a-t-il différents points, différents sujets sur lesquels vous êtes susceptible? Oui ? Non
(44) Est-ce que les autres vous considèrent comme quelqu'un qui est plein de vie? Oui ? Non
(45) Est-ce que vous vous sentez souvent désapointé? Oui ? Non
(46) Vous considérez-vous comme un individu bavard? Oui ? Non
(47) Avez-vous des périodes d'agitation ou d'impatience telles que vous ne pouvez rester assis longtemps sur une chaise? Oui ? Non
(48) Aimez-vous jouer des farces aux autres? Oui ? Non

Chapter 13

SOME EFFECTS OF CARISAPRODOL ON PAIN REACTIVITY

R. LYNN, Ph.D. and H. J. EYSENCK, Ph.D.*
(University of Exeter and University of London)

The experiment reported here was designed to test certain hypotheses relating to the effects of a new drug, Carisaprodol, on pain reactivity and various other psychological test responses; we were also interested in discovering how these responses would be related to personality. Our predictions were made on the assumption that Carisaprodol was acting as a C.N.S. depressant, an assumption made reasonable because of the close affinity of Carisaprodol to meprobamate (Berger *et al.*, 1959; Miller, 1959).

Subjects

Thirty subjects were tested in all; these were student volunteers at the University of Exeter. Each subject was tested three times: (a) under drug conditions, having been given a dose of 350 mg of Carisaprodol one hour before the beginning of the test; (b) under placebo conditions, having received a placebo indistinguishable from the drug; and (c) under "no drug" conditions. Five subjects were randomly selected to be tested under each of the six possible sequence conditions of a, b, and c.

Method

The subject was seated in an arm chair, his palms were cleaned with surgical spirit and the electrodes of the galvanometer were fixed to each palm with electrode jelly. Two minutes were allowed to elapse for the reading to become steady, and the resting level was then recorded (RL) (score in microamps). He was then told that his reaction to pain was going to be investigated and he was shown the thermal heat dolorimeter (Beecher, 1959) which stood immediately next to his chair. He was told that he would be asked to place the under side of his wrist on top of the aperture and the heat would be switched on. He was to keep his wrist on the aperture until he felt the prick of pain and then he should remove it. The intensity of the heat was set at 176 watts and pain was typically felt after about 6 sec. Two measures were taken from the PGR; the Anxiety Reaction

* We are indebted to the Wallace Laboratories for a grant which made this study possible, as well as for supplies of Carisaprodol ("Soma".)

(A) is the amount of increase in conductance as measured by the increase in microamps on the galvanometer scale, from the resting level to the reading when the subject places his wrist on the aperture of the thermal heat apparatus. The second measure was the Pain Reaction (P), the reading when pain was felt minus the resting level.

The subject was then instructed to lie back in the arm chair and close his eyes and his "orienting reflex" was extinguished. The stimulus was a mallet striking a block of wood; this stimulus was presented at 30 sec intervals until there was no PGR for three successive trials. The score recorded was the number of trials before the orienting reflex was extinguished (E).

He was then tested on the rotating spiral test (S) with 30 sec fixation (Eysenck, 1957). Finally he was tested for grip persistence (Costello and Eysenck, 1961). His maximum grip on the dynamometer was taken first (GM) and he was then asked to hold the pointer at half his maximum for as long as he could. His-persistence score was recorded in seconds (GT). In addition to these tests, each subject was given the Maudsley Personality Inventory (Eysenck, 1959).

Predictions

No prediction was made with respect to the resting level, as this measure was introduced merely in order to obtain a baseline from which to measure the changes in conductivity expected to result from the stimuli used to obtain scores A and P. We anticipated that the drug would *lower* the Anxiety and Pain responses, would *speed up* Extinction, *decrease* the duration of the spiral after-effect, and *increase* persistence.

Results

Mean scores are given in Table 1. It will be seen that with the exception of the persistence test all our expectations are, in fact, borne out. It is interesting to note that placebo reactions are intermediate between Drug and No Drug conditions; it is almost as if the placebo under these experimental conditions acquired some of the properties of the drug used. This is possibly due to the presence of certain features in the experiment which permit conditioning to take place, by pairing drug administration with depressant effects *before* the placebo is administered; such conditioning is, of course, only possible in certain groups of subjects, not in those in whom the placebo trial precedes the drug trial. An experimental investigation of this possibility would be of considerable interest.

Analyses of variance were carried out on all the data, and analyses of covariance on A and P, with Resting Level held constant. Furthermore, transformations of the skin resistance data were undertaken along the lines indicated by S.B.G. Eysenck (1956). These analyses will not be reported in detail as in none of them did the results achieve an acceptable degree of significance. It would appear reasonable to conclude that while Carisaprodol appears to have the predicted effects on all but one of the tests used, the dose chosen was too slight to make these effects sufficiently

TABLE 1
Scores

Test	*Drug*	*No Drug*	*Placebo*
Resting Level	32·77	29·73	31·33
Anxiety Reaction	4·63	5·27	4·50
Pain Reaction	9·37	11·27	10·20
Extinction	8·70	11·13	10·30
Spiral After-Effect	11·90	13·13	12·67
Grip; Maximum	65·63	66·03	65·27
Grip; Persistence	39·73	44·27	43·43

strong to overcome the very great variability in response to the tests which characterized our sample.

TABLE 2

N × RL_1	= —0·1799	E × RL_1	= 0·11998
RL_2	= 0·0587	RL_2	= 0·1308
(−) RL_3	= —0·1570	(+) RL_3	= 0·3677
N × A_1	= 0·2197	E × A_1	= 0·0013
A_2	= 0·2557	A_2	= —0·3070
(+) A_3	= 0·0702	(−) A_3	= —0·1230
N × P_1	= 0·0493	E × P_1	= —0·1387
P_2	= 0·3708	P_2	= —0·3372
(+) P_3	= 0·1365	(−) P_3	= —0·1757
N × Ex_1	= —0·1630	E × Ex_1	= 0·1971
Ex_2	= —0·3517	Ex_2	= 0·0808
(−) Ex_3	= —0·1388	(−) Ex_3	= —0·1014
N × S_1	= 0·0839	E × S_1	= —0·3373
S_2	= 0·2100	S_2	= —0·2994
(+) S_3	= 0·1863	(−) S_3	= —0·2453
N × GM_1	= —0·1770	E × GM_1	= —0·0222
GM_2	= —0·1065	GM_2	= 0·0087
(?) GM_3	= —0·1561	(?) GM_3	= —0·0007
N × GT_1	= —0·2152	E × GT_1	= 0·0631
GT_2	= —0·2616	GT_2	= 0·2817
(−) GT_3	= —0·2786	(+) GT_3	= 0·1732

It seemed relevant, therefore, to study the relationship between the experimental variables and personality dimensions N (neuroticism or emotionality) and E (extraversion–introversion), as determined by the M.P.I. (Eysenck, 1959), and as there were no significant drug effects, each person's questionnaire score was correlated with each test score three times, once for each successive administration of the test. (It is, of course, clear that to the degree to which the drug effects observed were *real* effects, this method would lower the observed correlations below the true correlations by confounding drug effects and personality.) The resulting correlations are given in Table 2, together with the direction of the relationship as it would be predicted from general theory; these predictions are given in brackets. They derive from the set of hypotheses developed in "*The Dynamics of Anxiety and Hysteria*", and extended in "*Experiments with Drugs*" (Eysenck, 1957, 1962). A (?) indicates that no prediction could be made.

Correlations of 0·362 and 0·464 are required to reach the 0·05 and the 0·01 level of P respectively, and relatively few of the observed values are as high as this. However, the three correlations in each set are nearly always identical with respect to sign, and may thus be taken as re-inforcing each other to some extent. In 11 cases out of 12 the observed correlations agree in sign with the predicted ones, taking the whole set of three into account; that is to say, either all three are in the predicted direction, or else two are in the right direction and the third one very slightly in the wrong direction. (Very slight, in this case, indicates values of 0·0013, 0·0087, and 0·0587.) In only one case (extraversion and extinction) is the direction of the forecast wrong; according to theory, extraverts should extinguish "orienting reflexes" more quickly, thus giving a negative correlation with number of trials fo extinction; the actual correlations are 0·1971, 0·0808, and —0·1014. All are insignificant, but only the last is negative, as required.* We may conclude from this survey of the observed correlations between personality and test scores that these are overwhelmingly in the predicted direction, but that the strength of the relationships indicated is below the level required for full statistical significance in most cases; if use had been made of the technique of "single-tail tests," then the number of significant correlations would have gone up decisively; Eysenck (1960) has given arguments against the use of this technique.

One prediction may require some discussion, viz. that relating to the PGR resting level. We have assumed that this is not, in fact, a true "resting level", as would be recorded, for instance, in sleep, but represents an autonomic "arousal" response to the situation of being in a strange room, full of apparatus, vaguely threatening and calling forth some degree of alertness. This "arousal" should adapt out in the course of several sessions

* It is possible that the positive correlation between extinction and extraversion, though not significant and contrary to theoretical expectation, deserves to be taken seriously. It is in line with a number of Russian studies showing that brain-injured subjects do not extinguish the orienting reflex as readily as do normals; also perhaps with the finding that animals higher in the phyletic scale extinguish these responses more readily (cf. Razran, 1961). It is to be regretted that so little work on this important response has been done in the West, and that there is almost none linking it with personality variables.

(and to some extent within each session), and when the figures are summed over subjects for the three successive sessions, we do, in fact, obtain an increase in resistance of some 15 per cent. (A similar adaptation is found with respect to the "orienting reflex", where there is a fall in number of responses of almost 50 per cent between first and third session.) From the fact that, theoretically, extraverts show quicker build-up of cortical inhibition (which is assumed to underlie adaptation, in so far as this is not due to peripheral factors), we would expect positive correlations between E and "resting level" resistance; this correlation should be highest on the third occasion, where subjects might be expected to have had enough time to adapt thoroughly. It will be seen that, indeed, the correlation for the third day is much the highest, as well as being the only significant one; this would seem to support the hypothesis. It is in line with this view thatin respect to extinction of the orienting reflex also, it is the correlation for the third day which is in line with prediction, while those for the preceding days are in the wrong direction. These points would probably repay more extended study.

SUMMARY

Thirty normal subjects have been given tests of pain reactivity, anxiety, "orienting reflex" extinction, spiral after-effect and persistence under conditions of "no drug", placebo, and 350 mg of Carisaprodol; they have also been administered the Maudsley Personality Inventory. Predictions were made with respect to (a) drug effects, and (b) personality correlates of test scores. Most of the predictions were, in fact, borne out by the results, but mostly at levels of statistical significance lower than 0·05. It is suggested that, in future work, larger doses of the drug may require to be used in order to obtain more clear-cut results.

Chapter 14

ETHYL ALCOHOL AND THE EFFECTS OF STRESS

J. EASTERBROOK*

Introduction

The physiological literature on ethanol can be described as confusing. The facts are distorted by errors, though there is enough redundancy among reports that some information comes through this noise (Easterbrook, 1961). Much the most important fault with the facts is the theory.

The psycho-pharmacology of ethanol (like that of other agents) has been influenced by the theoretical dichotomy of functions into depressant and stimulant. This has entailed difficulties because it has not been generally clear how sensitivity to discharge in the tissues of nervous assemblies may be related to activity by whole animals in psychological experiments. General theory has been inadequate as a source of standards of reference for psychopharmacology.

A point of general difficulty in the study of nervous efficiency which is reflected in the literature on ethanol is that the significance of rapid response to stimulation varies abruptly with the circumstances of test. When a single stimulus is effective and dominates an organism, it leads to intense experience and rapid impulsive action. Thus deprivations, assaults, intense stimulations and threats have been recognized to produce prevailing effects, known as "drive" (Hull, 1943) or general nervous arousal (Freeman, 1948), that have been described as activating or alerting because they facilitate such impulsive action. On the other hand, efficient and rapid adjustment to a multiplicity of disturbances can be achieved when the neural processes they initiate operate concurrently. To compound the confusion, it has become evident (Easterbrook, 1959) that rapidity of adjustment to stimulus complexes is impaired by the state of drive which produces rapidity in impulsive reaction.

Evidence to justify calling ethanol a CNS depressant refers primarily to rapidity of adjustment to stimulus complexes (Dale, Greenwood, Mellanby, Myers and Sherrington, 1938; Jellinek and McFarland, 1940). It seems to be true that subjects given relatively small doses of ethanol show poorer performance at complex tasks than do unemotional controls. At the same time, simple impulsive activity appears to be facilitated by ethanol. Indeed, it is known to be a stimulus to resting subjects. It causes positive response

*The writer is indebted to the committee of management of the Burden Neurological Institute, Bristol for the resources to carry out this investigation. The experiment described is part of a programme of work for the Ph. D degree in the University of London.

from chemo-receptors in the mouth and nose (Pfaffman, 1951; Diether, 1951; Kruger, Feldzamen and Miles, 1955; Lester and Greenberg, 1951), in blood vessels (Heymans, 1955; G. Liljestrand, 1953) and perhaps in the midbrain as well (von Euler and Soderberg, 1952; A. Liljestrand, 1953; Winterstein, 1961). It causes and facilitates depolarization of nerves directly (Gallego, 1948; Larabee and Pasternak, 1952; Grennell, 1957; Fischer, 1957; Ghosh and Quastel, 1954). It stimulates respiration (e.g. Dale *et al*, 1938; Gernandt, 1943; A. Liljestrand, 1953; G. Liljestrand, 1953; Holmberg and Martens, 1955; Loomis, 1952; Raffy, 1949) heart rate (e.g. Dodge and Benedict, 1915; Loomis, 1952; Holmberg and Martens, 1955) and endocrine activity (e.g. Eggleton, 1942, Kleeman, Rubin, Lamdin and Epstein, 1955; Perman, 1958; Kinzius, 1958; Santisteban, 1961). In its effects on both complex adjustment and impulse strength in quiescent subjects, ethanol operates like drive or general nervous arousal.

On the other hand, the emotional state of the subject apparently influences the effects of ethanol upon adjustment to stimulus complexes and also upon impulsive reactions. There is evidence to suggest that in emotion provoking conditions ethanol facilitates adjustment to complex situations. Such evidence comes primarily from experiments on the feeding behaviour of frightened animals (Dworkin, Raginsky and Bourne, 1937; Dworkin, Bourne and Raginsky, 1937; Masserman and Yum, 1946; Jacobsen and Skaarup, 1955; Conger, 1951), but its generality may be supported by studies on humans such as those of Ferrett, Barbut and Ducos (1951), and Vogel (1958). Under the influence of ethanol, ready responses to stimulus complexes are produced by subjects that otherwise behave inefficiently as a result of emotional states. In a similar way, according to evidence that is widely familiar (c.f. Easterbrook, 1961), ethanol reduces the intensity of impulsive reaction to intense drive-provoking stimuli so as, apparently, to reduce pain, fear and general stress. Here too, as in the case of adjustment to stimulus complexes, the effects of ethanol on subjects under weak stimulation are reversed in subjects under strong stimulation. Perhaps ethanol produces paradoxical effects of stimulus intensity (Pavlov, 1928).

The conception of ethanol as a stimulus contains hints for the explanation of these facts. It ought to act like other stimuli to precipitate the discharge and subsequent refractory phase of nerve cells it reaches. It ought, therefore, to facilitate impulsive response to all external stimulation and also to reduce the number of polarized cells available for subsequent external control and for recruitment by the more intense stimuli. It should act like noise. Finally, it should alter the normal relations of process intensity to stimulus intensity. It should do so in the fear–feeding conflict situation in such a way as to increase the intensity of the seeking processes and to reduce the intensity of the processes involved in fear.

The foregoing considerations have been formulated as hypotheses and put to test (Easterbrook, 1961). The hypotheses are:

(1) That increase in general nervous arousal due to increase in the intensity of prior stimulation will be associated with a reduction in the speed of response to adequate stimuli of low intensity in the presence of irrelevant stimulation.

(2) That small quantities of ethanol, internally, affect speed of response to adequate stimuli by facilitating response to weak stimuli and inhibiting response to strong stimuli.

(3) That small quantities of ethanol, internally, mimic the effects of irrelevant stimulation upon speed of response to relevant, adequate stimuli presented subsequently.

(4) That small quantities of ethanol, internally, affect the interaction between arousal and speed of response to adequate stimuli of low intensity in the presence of irrelevant stimulation, by : (a) increasing speed of response, in the case of subjects under the influence of strong drive-stimulation; and (b) reducing speed of response, in the case of subjects under the influence of weak drive-stimulation.

Method

The action selected for study was escape from electric shock. Differences in drive or arousal were produced in two ways: by preconditioning at different intensities of shock, and by varying the intensity of the irrelevant stimulus, white light. The use of aversive behaviour gives strong test to the hypothesis that the presence of the irrelevant light stimulus will alter the normal effects of drive.

Four intensities of relevant stimulus (SR) and four intensities of irrelevant stimulus (SI), in all their sixteen combinations, were presented to 16 subgroups from each of 3 major groups trained with different intensities of shock (ST) to produce three levels of relevant drive, arousal or stress. To observe the effect of a small quantity of ethanol on response in each condition doubled the required number of matched groups to a total of $(4\times4\times3\times2 =)$ 96 groups. Four animals were used in each group.

The intensities of shock for training were selected to produce relatively low emotion. The lowest intensity (60v) was 10 V higher than one which had been found inadequate to produce escape within 2 min. The highest intensity (100 V) was 50 V lower than one which had been found to make the escaping activity persist during inter-trial intervals.

The total of 4 rats was built up by running serially through the test conditions in four different sequences, duplicated daily. During sequences 1 and 3, ethanol was administered in morning tests, water in the afternoons, and the alternate order was followed for sequences 2 and 4. The four sequences were arranged in pairs to balance (as between extremes of stimulus intensity) any effects of order or time of testing, such as odours in the experimental chamber, prior disturbance in the stock room, weather and the condition of the apparatus. The four testing sequences are shown in Table 1.

The experimental chamber consisted of a space $6^1/_2$ in. $\times$ $6^1/_2$ in. $\times$ $5^1/_2$ in. (wide) in a box 13 in. $\times$ $6\ ^1/_2 \times 5^1/_2$ in. that had a metal cover for the opening at the top through which the rat was introduced. At one end of the box, a reflector carrying three light sources was separated from the experimental chamber by a frosted Perspex panel and a vacant space. The vacant portion of the box was divided from the experimental chamber by $^1/_{16}$ in vertical rods, set $^1/_2$ in. apart. Two side walls were covered by metal plates.

TABLE 1

Rata of test conditions showing the four sequences followed in serial testing

Number	*Training trials Relevant Stimulus Intensities (ST)*			*Testing trials Relevant Stimulus Intensities (SR)*				*Irrelevant Stimulus Intensities (SI)*			
	60 V	*70 V*	*100 V*	*60 V*	*70 V*	*100 V*	*150 V*	*0*	*2*	*10*	*312½**
1	1	2	3 (R)	1	2	3	4 (W)	1	2	3	4 (D)
2	3	2	1 (R)	4	3	2	1 (W)	4	3	2	1 (D)
3	3	2	1 (W)	4	3	2	1 (D)	4	3	2	1 (R)
4	1	2	3 (W)	1	2	3	4 (D)	1	2	3	4 (R)

(R) The parenthesized letters show the rate of rotation, by rat by day or by week.

* Light intensity in exposure meter units (Weston, Master III).

The chamber floor was a series of rods which could be electrified. When the shocking circuit was closed, these, the vertical rods and the side plates were each made electrically positive in random order for 25 msec once every 0·6 sec, the others serving as ground. Thus a rat in contact with only two electrodes had brief shocks during a twelfth of the time before it escaped. One with four points of contact was shocked for a sixth of the time.

At the end of the chamber opposite the light source was a movable plate of bright metal filling the available space and balanced against a switch to break electric circuits to grid and lamps. To escape the rat had to move this panel, as it easily did by standing with two paws on it or by pressure with its tail. This panel was never electrified. Above it, let into the lid of the box was a $^1/_{16}$ in. hole permitting entry of a small amount of room light.

On a shelf near the shock box, an electric fan produced a low ambient noise to mask the operator's movements.

The operator's control panel had means for selecting stimulus intensities, for initiating the stimuli and for resetting the entire switching and timing system. On each trial, stimulus levels having been selected, the operator had (a) to reset the system, (b) to press the light-starting switch, and (c) one second later to press the shock-starting switch. An electronic timer, zeroed at (a), began counting at (b) and stopped when the rat's switch was thrown, so that it recorded the time in seconds (to three decimal places) between pressure on the light-starting switch and completion of the rat's reaction. The tenth of seconds dial on this timer served to cue the operator in producing the one second inter-stimulus interval.

Three hundred and eighty-four rats served as subjects, being matched for strain (Wistar, albino, Glaxo) for sex (female) and for weight (140–160 g). They were randomly assigned to the conditions specified in the test rota.

A statistical procedure is mentioned below for supporting the assumption of the biological equivalence of the groups so composed.

All subjects were adapted to solitude in a darkened chamber by being kept for 18—23 hours just prior to testing in a cage that had been modified to resemble the experimental chamber in size and material. All were fed and watered *ad lib* up to the moment of testing.

Fifteen minutes before its test, each subject was given 1 ml of liquid by stomach tube and returned to its adaptation cage. For half the animals, this liquid was pure distilled water, but for half of them, twenty per cent of the water by volume had been replaced with ethyl alcohol, this dose being calculated at 1 mg of ethanol per gramme of average rat.

Observations were recorded by two different operators,* using the same equipment with common instructions. One made the morning observations while the other operated in the afternoon. Each was trained during pilot studies (a) to reproduce accurately the 1 sec interval between onset of stimuli, and (b) to administer the liquid by stomach tube smoothly (so that less than 20 sec was needed for the total operation beginning and ending with the rat in its adaptation cage).

The routine of testing days started with normal cleaning and feeding operations in the stock room which did not directly involve the experimental subjects. Half an hour after this work had ended, the morning experimenter injected his first rat, cleaned and polished the electrodes, started the masking noise and tested the equipment. The chamber was cleaned again after testing. An hour later the same procedure was repeated. The adaptation cages were then emptied and restocked for use on the following day.

Each rat was placed in the test chamber 15 min after entubation and allowed to explore it until she had pushed the movable panel once or until 2 min had elapsed. Six training trials were then given, with 2 min between the end of one and the beginning of the next. These were followed at the same rate by six test trials. Each rat, therefore, spent approximately 25 min in the test chamber.

For each rat, the operator recorded the conditions of each test and the time between light on and shock off, which was, of course, one second greater then the time the shock circuit was closed.

Analysis

Fourteen records in the ethanol sample and eighteen in the water sample were replacements for failure to meet fairly loose standards of consistency that are defined in detail in the original report (Easterbrook, 1961).

The raw times were transformed to remove the characteristic skew in distributions of reaction times. The transformation, selected by trial on the times for the first trial, was $100\sqrt{\frac{1}{t+1}}$ which of course is $100\sqrt{\frac{1}{RT+2}}$ because the measured time was 1 sec greater than the escape time (RT).

*The assistance of Mrs. Rita K. Carpenter is gratefully acknowledged.

The experiment yielded 4608 scores for speed of escape from shock. Thus, 4608 distinct factors might be required to account for the total variance in scores. It is the task of analysis to discover whether a more limited number of distinctions can serve this purpose to a useful degree, and if so to specify them.

The data fell conveniently into two sets of ($2\times6\times192$) 2304 scores, for both the training period (T 1—6) and the test period (T 7—12). Both sets of data were analysed in three stages:

(1) Simple analysis of variance, (a) to check that sample size was sufficient to produce the required compensation of random influences, and (b) to assess the importance of all the controlled variables, such as trials.

(2) Analysis of covariance and computation of least squares regression equations, (a) to smooth the differences between conditions in order further to support the assumption of biological equivalence of the test material, and (b) to distinguish significant from chance variations about the mean.

(3) Analysis of the integrated scores derived from the functions differentiated in (a), in order to assess the experimental hypotheses.

The details of the procedure used for smoothing and analysis of covariance are as follows:

(1) The voltage readings corresponding to the different values of shock stimuli were transformed into logarithms (base 10) for use in regression equations. The effectiveness of energy changes as stimuli is closely approximated by a log function of the physical values of these changes. The scale of irrelevant stimulus intensity is NOT a simple log. conversion of meter readings, however, but has an arbitrary component because there is no meaningful log. for zero energy change. The three positive values of light stimulus are indeed log. meter readings, but they were regarded as points on an arithmetic scale of stimulus intensity, which would have an arithmetic zero corresponding to the "no light" condition. This effectively codes the zero light stimulus as equivalent to an energy change of 0·1 units on the exposure meter scale. The empirical utility of this procedure was checked against the experimental data, first visually for simplicity of form of the relation between SI and speed of escape, and subsequently by an estimate of the error of fitting speed scores to these values. As will be seen below, a total sum of squares of 58,444 with 432 d.f. was attributable to the effects of SI. When the influence of SI was expressed in regression equations, however, a sum of squares of 4,163 was accounted for with eight degrees of freedom. The error of fit, then, is an MSV of 128·0, which is to be compared with the residual term of 132·9 in Table 3b. Evidently this somewhat arbitrary treatment of the transformation problem produced a scale which adequately approximated the effectiveness of the lights as stimuli. The values of the photic and the shock stimuli are shown in Table 2, which also displays the constants derived from them for use in regression analysis.

(2) The most significant independent variate was selected by reference to the variance analysis.

(3) Holding all other classifications constant, regression equations of the form $Y = a+b_1k_1(X)+b_2k_2(X)$ were then calculated for each cell. This form of equation was employed in place of the familiar $Y = a +$

TABLE 2

Values of the experimental variates as measured and as converted for use in analysis of covariance

Variate	*Initial Reading*	*Log/10 Reading* ($= X$)	k_1 ($= X - \bar{X}$)	k_2*
ST	60 V	1·7782	−0·9062	
	70 V	1·8451	−0·0293	
	100 V	2·0000	0·1256	
	$\bar{X}$	1·8744		
SR	60 V	1·7782	−0·17165	0·015957
	70 V	1·8451	−0·10475	−0·006442
	100 V	2·0000	0·05015	−0·023950
	150 V	2·1761	0·22625	0·014435
	$\bar{X}$	1·94985		
SI**	0·1 (×10)	0·0000	−1·699	1·522
	2·0 (×10)	1·3010	−0·398	−1·378
	10·0 (×10)	2·0000	0·301	−1·540
	312·5 (×10)	3·4949	1·796	1·396
	$\bar{X}$	1·699		

ST = Training Stimulus Intensity

SR = Relevant Stimulus Intensity

SI = Irrelevant Stimulus Intensity

* The k_2 constants are substitutes for $(X - \bar{X})^2$, calculated by solution of simultaneous equations which ensure that second order regression coefficient will be independent of the first.

** Measured with a photographer's exposure meter, calibrated in ASA units. See para (1) above on the rationale of conversion.

$+ b_1(X - X) \mp b_2 (X - X)^2$ in order to ensure the independence of the successive terms of the equation (Mather, 1951). The effective difference between the two formulae is a correction to $(X - \bar{X})^2$ to produce this orthogonality. The k_2 constants which do this are shown in Table 2 above.

Thus one large table of 576 total speed scores was replaced first by three smaller tables each containing an equal number of regression constants, a, b_1 and b_2.

(4) The material in these emergent tables was then examined in two stages with two further tables in place of each: (a) one showing the averages through trials of each constant, and (b) one showing the rates of change (b_1) with trials of these constants

(5) Calculations were then made of the variance in response speed (Y) represented by the regression constants in each derived table. This total variance was apportioned in a conventional way among and between the

various categories remaining in each table, and the significance of distinguishing between those categories was estimated.

(6) For each variate that emerged as significant from this analysis, calculations were made to test whether the number of constants might be further reduced by additional use of regression equations. This involved computations of the variance in Y encompassed by second order regression equations. To this end the conventional formulae were employed serially. Thus the variance in Y attributable to a b_1 coefficient may be calculated as $b_1^2 \times S(kX)^2$ (Mather, 1951). But $b_1{}^2$ is the variance of the coefficient b_1. Hence, when a second order constant b' is fitted to a matrix of b_1 coefficients, the variance in b_1 attributable to this second order coefficient is, conventionally, $b'^2 \times \times S(kX')^2$. This variance in b_1 is less than the corresponding variance in Y by a factor of $S(kX)^2$. Accordingly, the variance in Y attributable to a regression equation for b_1 is simply $b'^2 \times S(kX)^2 \times S(kX')^2$.

(7) All significant regression constants were collected and arranged in equations for predicting speed of escape from a knowledge of the experimental variates. These equations were then solved to obtain smoothed speed scores for each cell in the original matrices. These matrices of smoothed scores have a total variance equal to the total of the contributions of each constant in each equation, the various terms being algebraically independent.

This procedure yields functional relations between speed of escape and stimulus intensity with the effects of repetition of events and of other sources of stimulation held constant and specifiable. These relations represent the effectiveness of the stimuli in various combinations. They define surfaces that differ significantly from one another so that the final integrative stage of analysis can be pursued without heed to chance. They constitute experimental information.

Observations

The distributions of variance in speed scores among the various sets of conditions in water-treated, the ethanol-treated and the combined samples are given in Table 3 for both phases of the experiment.

It can be seen in Table 3 that in the stress-training phase of the experiment, significant proportions of the total variance were associated with the classification of shock stimulus intensity (ST) and of trials, without regard to the chemical condition of the subjects. On the other hand, the variance associated with intensity of shock was significantly greater in the water-treated than in the ethanol-treated sample; that associated with interaction between trials and shock level was considerable, but not significant when divided equally among the ten pertinent degrees of freedom.

In the test phase of the experiment, significant variance was again associated with distinctions between trials and relevant (shock) stimulus intensity (SR). Here, too, a significant interaction was found between the intensity of this relevant stimulus and chemical condition, but in this case, the effect of stimulus intensity was greatest for the ethanol-treated subjects.

As anticipated, the data of the test phase of the experiment revealed significant evidence of stimulus interaction. The variance associated with distinctions between relevant stimulus intensity was significantly different

TABLE 3(a)

Mean square variance estimates and degrees of freedom D.F. Thereof for speed of escape during training trials

Index	(*D.F.*)	*Water*	*Ethanol*	*Communal*	*Differential*
Mean	(1)	3,029,798	3,056,319	6,086,080	37
(Total Variance)	(1151)	(176,148)	(175,928)	(352,113) †	
Main Effects					
Stimulus Intensity (ST)	(2)	5,084*	2,290*	6,881*	474*
Trials (T)	(5)	688*	1,275*	1,896*	49
Interactions					
ST × T	(10)	44	179	140	84
Residual	(1134)	142·9	144·0		143·5

The table displays results of partition of the total variance within the water, the ethanol and the combined samples. The classification of the latter as communal and differential effects is conventionally set forth as main effects and interaction (with chemical treatment).

Asterisks indicate variance estimates yielding MSV's that significantly exceed the relevant (residual) error terms at the 0·05 level of confidence.

† This total has (2 × 1151 =) 2302 degrees of freedom.

TABLE (3b)

Mean square variance estimates and degrees of freedom (D.F.) thereof for speed of escape during training trials

Index	(*D.F.*)	*Water*	*Ethanol*	*Communal*	*Differential*
Mean	(1)	3,625,451	3,580,042	7,205,422	71
(Total Variance)	(1151)	(159,765)	(163,011)	(322,848) †	
Irrelevant St. Int. (SI)	(3)	47	88	112	23
Relevant St. Int. (SR)	(3)	2,093*	4,812*	6,246*	658*
Training St. Int. (ST)	(2)	297	25	247	76
Trials (T)	(5)	243	313*	325*	231
SI × SR	(9)	337*	250	418*	168
SI × ST	(6)	200	171	135	236
SI × T	(15)	74	85	80	78
SR × ST	(6)	251	95	238	108
SR × T	(15)	144	86	131	99
ST × T	(10)	99	155	125	129
SI × SR × ST	(18)	295*	199	298*	258*
SI × SR × T	(45)	96	100	102	126
SI × ST × T	(30)	69	85	87	67
SR × ST × T	(30)	104	111	124	123
SI × SR × ST × T	(90)	112	130	144	115
Residual	(864)	135·0	130·9		132·9

* † See Notes to Table 3(a).

for different intensities of the irrelevant stimulus (SI). This value, in turn, differed significantly for different intensities of stress or training stimulus, and to a significantly smaller degree in the ethanol sample. Again, however, none of the interactions with trials was significant when the total variance was divided equally among the relevant number of degrees of freedom.

The analysis of variance may be supposed to have shown that the sample of 4 rats per cell was large enough to reduce the effect of random differences between animals to a level permitting display of the influence of the experimental variates. It has also shown that process interaction did, in fact, occur. On this evidence, no further replications were sought and the investigation moved instead to detailed examination of the manner in which the various influences had operated.

Analysis of covariance in the data from the training phase of the experiment showed that the 26,187 units of variance in speed of escape which were associated with 35 degrees of freedom in Table 3 can be adequately represented with four empirical constants: a mean of 51·46, a rate of change with trials of 1·18 and two rates of change of speed with stimulus intensity of 31·67 and 18·62 for the water-treated and ethanol-treated groups respectively. These four constants account, in all, for 22,776 units of variance, the remaining 3942 units being taken up by the 31 residual degrees of freedom. The mean square variance (MSV) of this error of fit is 127·73, compared to the error MSV of 143·46 for the whole of the data in this phase of the experiment.

These results show that during the first six episodes of escape from shock in the absence of other influences:

(1) Speed of escape increased with increase in the intensity of shock.

(2) Ethanol reduced the rate of increase in speed of escape with increase in the intensity of shock.

The results of analysis of covariance in speed scores with experimental conditions in the test period are summarized in Table 4. This table shows separately the regression constants found to represent the non-random variations in speed in the records of the water-treated and the ethanol-treated groups, as well as the variance attributable to each expression and to the average of, and difference between, them. Further detail on the derivation of these equations may be found in the original report (Easterbrook, 1961).

Table 4 is divided vertically into four sections, each referring to analysis of a different sort of influence. The two upper segments refer to sums through trials and the two lower segments to the effects of trials. The uppermost segment of each pair refers to influences affecting speed scores at all intensities of present shock (SR), and the lower one refers to influences on the relation between performance and the intensity of present shock (i.e. to interactions with SR).

Departures from the grand mean due to influences set out in the uppermost segment of Table 4 are represented graphically in Fig. 1. They show that speed of escape from electric shock of average intensity was not directly affected by ethanol and that:

TABLE 4

Summary of regression equations relating speed scores for escape to conditions of test phase of experiment

*Regression Constants**	*Regression Coefficients*		*Variance Attributable to Expression*			
	Water	*Ethanol*	*Water*	*Ethanol*	*Communal*	*Differential†*
a	56·10	55·75	3,625,451	3,580,042	7,205,422	71
*k*ST	7·680	2·475	586	61	256	391
*k*ST*k*SI	−4·685	5·388	347	459	4	802
*k*SR	14·698	22·573	5,857	13,827	18,830	854
*k*SR*k*ST	−48·439	−29·751	549	207	715	41
*k*SR*k*ST*k*SI	44·910	−13·794	750	71	180	641
*k*SR*k*STk_2SI	17·708	17·708	156	156	313	0
k_2SR	34·300	−52·914	365	869	54	1,181
k_2SR*k*SI	−36·536	−36·536	660	660	1,320	0
*k*T	0·164	0·482	91	779	699	171
*k*T*k*ST	−1·421	4·249	59	523	115	467
*k*T*k*SR	−1·372	−1·372	159	159	318	0
*k*T*k*STk_2SR*k*SI	−111·151	−111·151	153	153	307	0
*k*T*k*STk_2SRk_2SI	−133·658	−133·658	298	298	596	0
		Sums	3,635,484	3,689,264	7,229,129	4,619

* The constants are the matrices composed by the indicated products of the values shown in Table 2.

† More conventionally read as interaction with ethanol. See note toTable 3.

(1) In the absence of irrelevant stimulation, speed of escape was positively related to the intensity of shock used during training in the prior trials (ST), i.e. to the intensity of the relevant source of drive.

(2) Addition of irrelevant stimulation affected speed of escape (a) on the average by reducing the rate of increase in speed with training stimulus intensity, and (b) in proportion to its intensity, by facilitating the escape of animals trained with weak shock (as a drive stimulus should), and inhibiting the escape of animals trained with strong shock.

(3) Treatment of the animals with ethanol had the effects (a) in the absence of irrelevant stimulation, of reducing the rate of increase in speed with training stimulus intensity, and (b) in the presence of irrelevant stimulation, of reversing the effect on speed of escape of increase in the intensity of either ST or SI when the other is held constant.

The calculations in the second segment of Table 4 refer to the parameters of effectiveness of the relevant stimulus (SR). They show that diffeernces

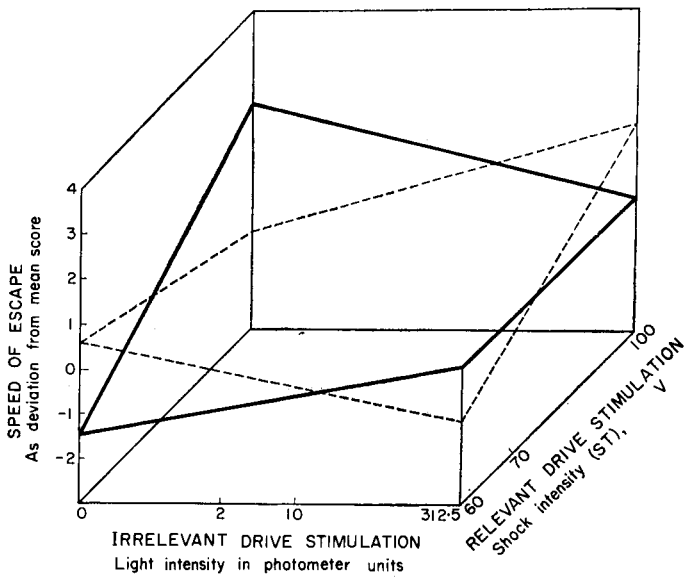


FIG. 1.

in the intensity of SR account for the greatest part of the non-random variance in the experiment and that:

(1) In the absence of irrelevant stimulation, (a) speed of escape increased with increase in the intensity of present shock, and (b) the rate of increase in speed of escape with intensity of present shock was reduced by increase in the intensity of prior shock (ST)

(2) Addition of irrelevant stimulation (SI) affected speed of escape in proportion to the intensity of irrelevant stimulation by (a) offsetting the effect of intensity of prior shock (ST) on the rate of increase of speed with intensity of present shock (SR) and simultaneously (b) increasing the rate of increase of speed with intensity of present shock

(3) Treatment of the animals with ethanol had the effects of (a) increasing the rate of increase of speed with intensity of present shock and simultaneously (b) enhancing the reduction by irrelevant stimulation of the tendency of prior shock to displace present shock as a determining variable.

The relations between repetitions of stimulation and speed of escape are summarized in the lower segments of Table 4. These results show that:

(1) Without influence by chemical treatment the effects of repetitions of stimulation were: (a) a steady increase in speed of escape; (b) a reduction in the rate of increase in speed of escape with increase in present shock; and (c) a reduction in the interactive effect of prior shock (ST) and present irrelevant stimulation (SI) upon the rate of increase in speed of escape with intensity of present shock (SR).

TABLE 5

Time in seconds from onset of shock required for rats treated with water to press switch and turn off shock under indicated conditions

Training Trials

T/ST	*60 V*	*70 V*	*100 V*
1	2·84	2·42	1·63
6	1·79	1·50	0·93

Test Trials

T SI	*ST/SR*	*60 V*	*70 V*	*100 V*	*150 V*
7 Zero	60 V	1·91	1·94	1·56	0·65
	70 V	1·55	1·65	1·44	0·66
	100 V	0·90	1·12	1·19	1·52
Low	60 V	1·77	1·85	1·52	0·60
	70 V	1·59	1·60	1·33	0·69
	100 V	1·21	1·10	0·95	0·92
Mod.	60 V	1·75	1·73	1·38	0·68
	70 V	1·62	1·55	1·24	0·74
	100 V	1·35	1·13	0·95	0·89
High	60 V	1·80	1·32	0·87	1·13
	70 V	1·72	1·40	0·99	0·92
	100 V	1·54	1·58	1·29	0·51
12 Zero	60 V	1·51	1·82	1·43	0·65
	70 V	1·27	1·42	1·36	0·71
	100 V	0·81	1·02	1·21	0·86
Low	60 V	1·64	1·44	1·01	0·74
	70 V	1·37	1·35	1·16	0·78
	100 V	0·85	1·13	1·38	0·87
Mod.	60 V	1·56	1·36	1·02	0·79
	70 V	1·39	1·31	1·10	0·82
	100 V	1·02	1·14	1·28	0·90
High	60 V	1·09	1·20	1·22	0·86
	70 V	1·31	1·23	1·06	0·90
	100 V	1·94	1·30	0·73	0·98

TABLE 6

Time in seconds from onset of shock required for rats treated with ethanol to press switch and turn off shock under indicated conditions

Training Trials

T/ST	*60 V*	*70 V*	*100 V*
1	2·58	2·35	1·87
6	1·61	1·45	1·10

Test Trials

T SI	*ST/SR*	*60 V*	*70 V*	*100 V*	*150 V*
7 Zero	60 V	1·67	1·47	1·05	0·62
	70 V	1·87	1·65	1·17	0·69
	100 V	2·40	2·12	1·50	0·87
Low	60 V	1·92	1·67	1·11	0·54
	70 V	2·03	1·68	1·11	0·71
	100 V	2·33	1·72	1·09	1·17
Mod.	60 V	2·11	1·70	1·04	0·60
	70 V	2·14	1·68	1·04	0·75
	100 V	2·22	1·62	1·03	1·17
High	60 V	2·71	1·60	0·70	0·98
	70 V	2·44	1·61	0·84	0·93
	100 V	1·85	1·64	1·21	0·79
12 Zero	60 V	1·49	1·34	1·06	0·78
	70 V	1·45	1·33	1·02	0·67
	100 V	1·45	1·29	0·93	0·57
Low	60 V	1·95	1·43	0·84	0·73
	70 V	1·67	1·33	0·87	0·74
	100 V	1·12	1·10	0·95	0·62
Mod.	60 V	2·07	1·48	0·84	0·81
	70 V	1·74	1·33	0·83	0·76
	100 V	1·11	1·01	0·81	0·66
High	60 V	1·91	1·61	1·14	0·84
	70 V	1·78	1·33	0·83	0·82
	100 V	1·51	0·80	0·27	0·79

(2) In the ethanol group only, the effect of repetitions of stimulation was to increase the positive association between speed of escape and intensity of prior shock.

The 23 empirical constants in Table 4 represent 7,233,748 of the 7,528,270 units of variance among the 2304 speed scores obtained from the experiment. Ignoring the mean, thus, 28,326 units of variance are encompassed by 22 degrees of freedom in this table. The residual, with 2281 degrees of freedom, gives an M.S.V. 129·1, which may be regarded as the error variance for this phase of the experiment.

Not all of the entries in Table 4 are significant at the 0·05 level of confidence. There are two reasons for this. First, the table shows as individual M.S.V.'s with one degree of freedom the contributions of terms from more complex expressions which have a broader basis collectively. Secondly, it sometimes happens that significant differences attributable to chemical condition arise when the relevant influence is insignificant in one of the treatment groups. The material in all parts of the tables is interrelated and, collectively, highly significant.

The average speed scores calculated from the equations for both the training period and the test period provide the basis for observations pertinent to the experimental hypotheses. Transformed to read as escape times, these scores are presented in Tables 5 and 6.

Two of the hypotheses about ethanol refer to impulsive reaction to stimulation. Their assessment depends on comparisons with the average speeds of escape by control subjects acting in the absence of irrelevant stimulation. These comparisons can be made directly from the material of Tables 5 and 6. They justify the assertions to be made below. Those which refer to the first test trial are illustrated in Fig. 2. It is evident that:

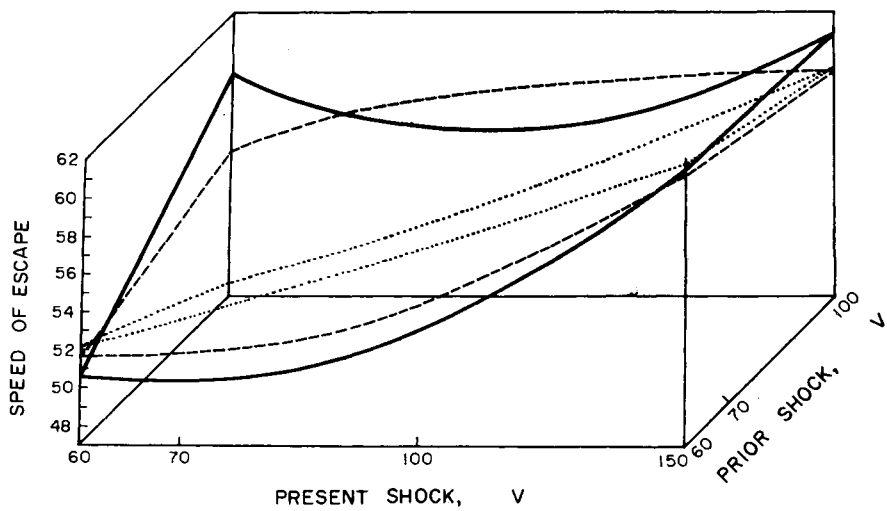


Fig. 2.

(1) During the first 6 episodes, speed of escape was facilitated by increase in the intensity of shock, facilitated by repetitions of stimulation and affected by ethanol (a) facilitatively when shock level was low and (b) inhibitively when shock level was high.

(2) During test trials, increase in the intensity of prior shock (a) facilitated escape on the first test trial when the intensity of present shock was weak, slightly inhibited escape when present shock was strong, and (b) reduced the rate of increase in speed with repetitions of stimulation.

(3) Ethanol altered the effect of prior shock during test trials so that, with present shock intensity either low or high, (a) in the case of subjects trained at low shock, it facilitated response on the first trial and reduced the rate of increase in speed with repetitions of stimulation, and (b) in the case of subjects trained at high shock, it inhibited response on the first trial and increased the rate of increase in speed with repetitions of stimulation.

(4) Introduction of irrelevant stimulation during test trials altered the effect of prior shock so that, with present shock intensity either low or high, (a) in the case of subjects trained at low shock, it facilitated response on the first trial and reduced the rate of increase in speed with repetitions of stimulation, and (b) in the case of subjects trained at high shock, it inhibited response on the first trial and increased the rate of increase in speed with repetitions of stimulation.

The two remaining hypotheses refer to speed of response to weak relevant stimuli in the presence of irrelevant stimulation. The escape times in Tables 5 and 6 can be used to make the necessary comparisons. Those referring to performance in the absence of irrelevant stimulation should be subtracted from those pertaining to escape in its presence. Negative values then show facilitation of escape by the irrelevant stimulus and positive values show inhibition.

To determine the effect of repetitions of stimulation on speed of response to weak relevant stimuli in the presence of irrelevant stimulation, comparisons of a similar sort are required. Using the escape times of Tables 5 and 6, subtraction of the difference (T 12—T 7) in times required in the absence of SI from the similar difference in its presence yields positive values when the irrelevant stimulation had the effects of either reducing escape time on the first test or increasing it on the last. By allusion to the fact that the irrelevant stimulus stood to the relevant stimulus as a conditional stimulus to an unconditional stimulus, these differences may be designated "contingent" inhibition, or facilitation.

When the necessary comparisons are made, it is evident:

(1) As illustrated in Fig. 3. with speed scores, the effect of increase in the intensity of prior stimulation from either the training stimulus or the irrelevant stimulus was to reduce speed of escape on the first test trial (although irrelevant stimulus intensity made little difference in the case of subjects trained with low shock).

(2) As illustrated in Fig. 4, repetitions of stimulation (a) facilitated escape under conditions in which irrelevant stimulation produced moderate facilitation or moderate inhibition of escape by control subjects on the

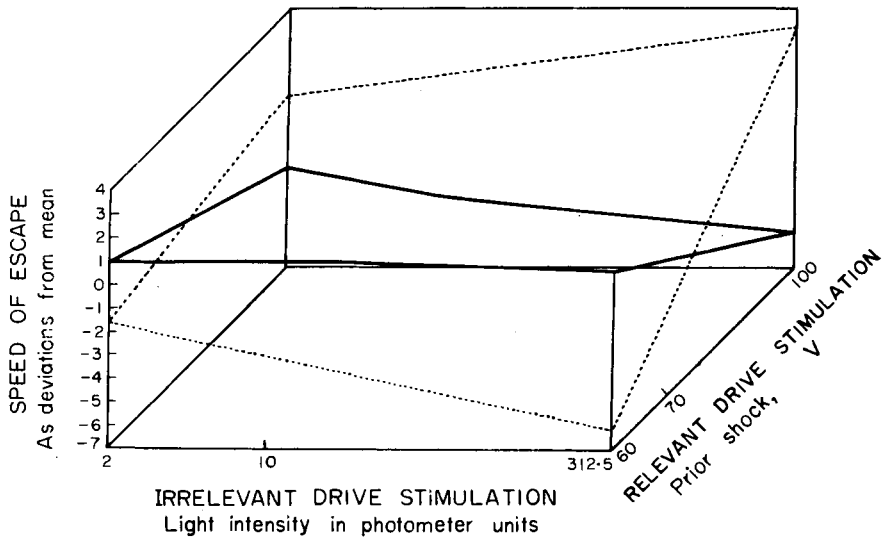


FIG. 3.

average trial, and (b) inhibited escape under conditions in which irrelevant stimulation produced minimal or maximal facilitation, or minimal or maximal inhibition of escape on the average trial.

(3) Ethanol (a) reversed the effect of increased intensity of drive stimulation from either source on speed of escape on the first test trial, as shown in Fig. 3, and (b) altered the relation between the effects of repetition of stimulation and the effects of irrelevant stimulation on the average trial, but (c) did not affect the influence of repetitions of stimulation on speed of escape.

Conclusions on Hypotheses

By observation it is now evident that:

(1) Increase in arousal due to prior stimulation was associated with a reduction in speed of escape from shock of low intensity in the presence of irrelevant light stimulation.

This statement elides a very slight discrepancy. The effect of increasing the intensity of irrelevant stimulation from low to moderate, when relevant drive was low, was to increase speed of escape on the first test trial. The extent of this effect is regarded as negligible, however, for it amounted to a mere 2/100ths of a second. Accordingly, the first experimental hypothesis is regarded as having gained substantial support in fact.

(2) Internally, 100 mg of ethanol per 100 g of average rat affected speed of escape from adequate stimuli facilitatively under the influence of low intensity drive stimulation and inhibitively under the influence of high intensity drive stimulation, but it *did not* generally facilitate escape from weak stimuli and inhibit escape from strong stimuli.

In view of this evidence, the second hypothesis must be rejected, although a modified version referring to the effectiveness of drive stimuli appears to be acceptable.

(3) Internally, 100 mg of ethanol per 100 g of average rat caused changes in escape from adequate stimuli, presented subsequently, which mimic those due to irrelevant stimulation.

The third hypothesis has accordingly found substantial support in this experiment.

(4) Internally, 100 mg of ethanol per 100 g of average rat affected the interaction between arousal and speed of escape from adequate stimuli of low intensity in the presence of irrelevant stimulation by facilitating escape by subjects treated to produce high arousal and by inhibiting it in the case of subjects treated to produce low arousal.

In the light of this experimental information, accordingly, the fourth hypothesis has found substantial support.

The Biological Action of Ethanol

Changing the level of description, the experiment has shown that ethanol, and indeed irrelevant stimulation too, generally reduced the influence of past events upon behaviour and enhanced the influence of present events. In human behaviour, it is just such a difference between sources of influence that has defined the concept of *extraversion.* Behaviour determined by the present situation is *extraverted,* by definition, while that which shows the greater influence of prior experience is *introverted,* by abstraction of definition. It will be recalled, of course, that the McDougall–Eysenck theory (McDougall, 1929; Eysenck, 1957) specifies that ethanol produces increased evidence of extraverted behaviour and reduced evidence of introverted behaviour. Data of the present study would be cited as illuminating the mechanism of extraversion, had they come from experiment on humans. Perhaps, despite the odd variety of anthropomorphism implied in doing so, these observations may be tendered for the same purpose.

Eysenck's propositions about ethanol in terms of inhibitory and excitatory potentials (1957) also find quite specific support in the present study, but only under limited conditions. Ethanol inhibited escape from weak stimuli in the presence of irrelevant stimulation after training with weak shock. These conditions may not severely restrict the generality of that hypothesis, however, for they were contrived to produce behaviour representing the use of cues by unemotional subjects.

The information arising from this experiment gives partial support to other hypotheses as well. Indeed, it is a remarkable thing about these results that at different places they provide evidence analogous to that required by such a variety of apparently discrepant psychological propositions about ethanol. Besides supporting the McDougall–Eysenck hypotheses, they show that, in certain circumstances, ethanol reduces the utility of weak stimuli, as Masserman and Yum declare (1946), reduces the effectiveness of intense drive as supposed by Ullman (1952, 1953) and perhaps by Conger (1951, 1956) and Miller (1951) too, reduces conflict and improves

performance as, for instance, Masserman and Yum (1946) and Vogel (1958) report, produces paradoxical effects of stimulus intensity (Pavlov, 1928), "depresses" and "disinhibits" as the classical conception of its nature asserted (Dale *et al.*, 1938). The only important generalization that gains no support from these data is the doctrine that "... the direct action of alcohol upon the nervous system is, in all stages and upon all parts of the system, to depress or suspend its functions; that it is, in short, from first to last a narcotic drug" (Dale *et al.*, 1938, p 42).

Simple, single-stimulus situations do not constitute good analogues of worldly situations. None the less, they are exactly correct for assay of the effects of a treatment on the strength of nervous impulse *in vivo*. For, they contain no stimuli to inhibit response, nothing to do so, indeed, beyond the normal resistances to activity which stimuli overcome. Accordingly, evidence that action is facilitated in such settings cannot be described as evidence of disinhibition as the classical conception of ethanol requires. The facilitative effect of ethanol on the arousal evoked by weak stimuli in this experiment cannot be explained as inhibition of inhibition. It is stimulation of the kind normally produced by increase in the intensity of prior stimulation (and it has similar effects on the effectiveness of relevant stimuli in the presence of irrelevant stimulation). The suggestion that ethanol makes these changes by acting as a stimulus is supported by evidence that it mimics irrelevant stimulation in this respect among others.

Although facilitation of simple impulsive action cannot be explained by supposing that a treatment depresses nerve sensitivity, the converse is not true. Depression can be explained by increased sensitivity or by increased stimulation. The necessary argument has already been made. It depends on the fact that nervous discharge produces a refractory period and on the presumption that ambient stimulation speeds up the rate of exhaustion by external events of the supply of polarized cells. Ethanol as ambient stimulation ought to displace other sources of arousal. Presumedly its evident effect in reducing both the facilitative and the inhibitive effects of intense drive-stimuli is due to such displacement.

The Effects of General Arousal

It appears to be generally true that increase in the intensity of nervous events in progress reduces the capacity of an organism to sustain overlapping processes. Without knowing the total capacity in advance, however, it will be simply a matter if luck of the first increment in the intensity of an irrelevant process should inhibit any specified reaction. Before the effects of capacity-reduction begin to show themselves, the known tendency of arousal to amplify the strength if dominant impulses ought to produce net facilitation (which, however, would be reduced by increase in arousal). This argument presumedly accounts for the fact that, with the relevant drive at its lowest, irrelevant stimulation actually facilitated response by the control subjects in this experiment. It inhibited the response of subjects treated with ethanol, hypothetically because of the added stimulation which that treatment entailed. Arousal is, therefore, conceived to have two effects:

amplification of process intensity and reduction of capacity to sustain overlapping processes.

If arousal has two effects, there ought to be two phases in its influence on response to stimulation. During the first phase, the intensity of all stimulated processes ought to increase until the system's capacity has been exhausted. During the second phase, thereafter, the intensity of some processes should continue to rise at the expense of others so that the total number of processes is reduced. Any operation which is dependent upon the joint effectiveness of the critical number of stimuli ought to break down after that point.

The foregoing argument may be illustrated by reference to the interesting observations which have been made about "contingent facilitation". Gains due to practice in the effectiveness of weak relevant stimuli in the presence of irrelevant stimulation define "contingent facilitation". When such measures were averaged for the conditions which produced different speeds of escape on the average trial in the presence of irrelevant stimulation, they yielded the curvelinear relations shown in Fig. 4. Now escape speeds on the average trial may be interpreted as measures of the intensity of the pertinent processes, so that negative measures indicate the intensity of process initiated by the irrelevant (inhibiting) stimulus and positive measures indicate the intensity of process initiated by the relevant (excitatory) stimulus. The striking coincidence of the relation each of these measures shows to contingent facilitation is the point of interest. Hypothetically it means that changes in arousal intensified both processes up to a level at which they persisted concurrently and contingent facilitation occurred. Beyond that point one process or the other was intensified while the other was inhibited so that they ceased to be concurrent, and contingent facilitation was no longer possible.

Psycho-physiological Method

In larger context, it may be thought that one of the principal contributions of this study lies in its demonstration of the sensitivity of speed scores to modification both by differences in stimulus intensity and by repetitions of stimulation. This sensitivity was such that the experimental results depend entirely upon assessments for each trial of the differences in speed of escape due to differences in the intensity of various stimuli. These results would have been obscured completely in any analysis that failed to make these differentiations. This is shown by, for example, the lack of a main effect of ethanol in the variance analysis. However, by making these differentiations both in theory and in method, the pharmacological concepts of stimulation and depression and the physiological concepts of facilitation and inhibition have been given specific operational definition and have been investigated in this study.

Another possible contribution to psycho-physiological technique is the method used in the experiment for assessing the availability of capacity to sustain overlapping processes without the use of complex behaviour. The animal is required to make rapid selective response to one type of

stimulus while being subjected to irrelevant stimulation. Such selective responses are made efficiently and rapidly only when processes evoked by the various stimuli can operate concurrently rather than consecutively. The use of irrelevant stimulation, accordingly, serves to distinguish between the rapidity of response which is due to strength of impulsive action and the rapidity of response which is dependent upon capacity for concurrent reaction to a number of stimuli. The effect of a treatment on both these determinants of efficiency can be assessed in a simple setting.

It is notable, too, that the major independent variables of this study were inadequate to produce the escape reactions used to externalize neural processes. These variables took effect by modifying the normal relations between speed of escape and the intensity of the stimulus which was adequate to produce escape. Statistically speaking, their effects were known entirely by these interactions. This may be another way of saying that the use of experimental cues is motivated; it is certainly another way of expressing the relationship between adequate and inadequate stimulation in the study of behaviour. Hull (1943) and others have taken the view that inadequate stimuli are rendered effective by drive, through some multiplicative process. What is suggested here is simply that the utility of inadequate stimuli can be displayed in behaviour only by interaction with the effects of adequate stimuli.

Finally, this study has demonstrated that an important principle, previously expounded in reference to introspective observations on humans, can be discerned in the behaviour of rats. This is the "Psycho-physical law" (Stevens, 1957). In engineering terms, it says that when the noise level rises, the intensity of events that will serve as signals must also rise. That it probably represents a fundamental characteristic of nervous systems will be evident to readers of Sherrington's work with spinal dogs (1906).

SUMMARY

Two major indicants of the efficiency of nervous systems in adjustment to outside disturbances are the strength of single stimulated nervous processes and the number of such processes that can persist concurrently and interact. That each may become manifest in rapid action in certain circumstances has been a source of confusion in psycho-physiology.

Drive or general nervous arousal, the prevailing effect of prior disturbance, is widely known to facilitate simple action. It has, therefore, been described in terms of "alerting" and "activating" nervous systems. Recently, also, it has been shown to impair performance that is facilitated by overlap of serially stimulated processes.

Ethanol, taken internally, has been classified as a nervous depressant. In special circumstances, it impairs performances that are facilitated by the overlap of serially stimulated processes. At the same time, according to recent literature, it evidently facilitates single impulsive acts.

An experiment is described to test the utility of this system of classification for reconciling apparently discrepant facts. It tests the separate and interactive effects of arousal and ethanol upon speed of escape by

rats in the absence and in the presence of irrelevant stimulation. Rapid escape in the latter case demonstrates capacity to respond to the relevant stimulus despite the effectiveness of the irrelevant stimulus. In the former case, rapidity of escape demonstrates the strength of single impulse.

General nervous arousal and internal ethanol are both shown to facilitate simple response to a single stimulus and to reduce capacity to sustain overlapping processes. Their effects interact. Ethanol enhances the effect of weak sources of arousal, reduces the effect of strong sources, and influences capacity to sustain overlapping processes as if that capacity were primarily and negatively dependent upon arousal; it is conceived as a stimulus.

Several general points are made about psycho-physiological methods as used in experiments of this type.

Chapter 15

THE INFLUENCE OF STIMULANT AND DEPRESSANT DRUGS ON THE CENTRAL NERVOUS SYSTEM

R. N. Gooch, M. B., B. S.

Introduction

Since antiquity mankind has used a variety of substances taken for the purposes of producing subjective effects of a more or less pleasurable kind, often for religious or magical purposes. Alcohol is a notable example, but caffeine, cocaine, opium and other plant derivatives have been similarly used at times in various cultures. Along with the recognition of these subjective effects went almost certainly a recognition of relatively specific behavioural effects associated with each of these substances; the deliberate usage in early times of such substances to produce changes in behaviour represents a primitive fore-runner of an important area in modern pharmacology.

To-day the pharmacologist has in his armamentarium a considerable range of drugs capable, by their action on the central nervous system, of exerting an effect on subjective experience and behaviour. The differences between these agents are fairly well delineated so that they may be categorized in a loose descriptive way by applying such adjectives as "sedative," "stimulant," "ataraxic" etc. with respect to the predominant action of the drug in question. Many of these psychotropic agents display the property of stimulating or depressing the functions of the central nervous system, in either a general or a more specific fashion.

Besides recognizing differences between individual drugs, differences of response to a particular drug can be observed from person to person. Whilst some of the factors involved in this individuality of response to drugs are obvious (e.g. in terms of age, bodily size, state of physical health, acquired tolerance etc.) many other factors remain comparatively obscure. One complex source, possibly, contributing to individual differences in drug response is personality, which has recently received much attention from investigators interested in the effects of drugs upon behaviour.

Whilst, on the one hand, development has been occurring in experimental psychology with the usage of drugs to investigate behaviour, other techniques have been developed by the physiologist interested in the effects of drugs upon central nervous system functioning. This has led, with respect to psycho-pharmacology, to a convergence of the interests of the psychologist and the physiologist. Whilst the psychologist is more interested in the molar aspects of behaviour in relation to drug response, some knowledge of the neurological and pharmacological aspects is essen-

tial, both for the formulation of economical experimental design and as an adjunct to the psychological interpretation of behavioural data. It is with these needs in mind that the following consideration of stimulant and depressant drugs is presented.

Consciousness, Activation and Electrophysiological Arousal

It may be said that the general central nervous system depressant drugs are a family of pharmacological agents which tend to produce a depression of the functions of the central nervous system manifested (depending on dosage, relative efficacy of the drug, and such variables as individual susceptibility) by sedation, soporific effect or unconsciousness accompanied by anaesthesia (Wilson and Schild, 1959). On the other hand, those drugs accredited with a non-specific central nervous system stimulant tend to produce behavioural manifestations of the opposite kind such as restlessness, insomnia and varying degrees of excitement (generally in terms of both gross motor behaviour and expressive display). Moreover, the two families of drugs tend to be pharmacologically antagonistic in this respect when administered concurrently. Broadly speaking we may consider both groups of drugs as tending to produce alterations in the level of consciousness.

Consciousness has been defined in terms of an "awareness of environment and of self" (Cobb, 1948) in which is implicit the concept of potentiality for a responsiveness to stimuli which is in some way appropriate to the nature of the stimulation. One approach to the problem of defining the level of conscious activity in an individual at a given time is in terms of a consideration of his ability to respond to a given stimulus or pattern of stimuli. A gross example of this approach may be found in the clinical tests used by the physician in determining whether an apparently unconscious patient is lightly stuperose or deeply comatose from the nature and appropriateness of his response (or lack of it) to the application of an ordinarily painful stimulus.

A useful way of considering level of conscious activity is the concept of "activation." This concept has been put forward by a number of people including Malmo (1959) who in a recent review regards the level of consciousness as a function of the degree of "cortical activation" which is attained in response to corticopetal impulses derived from lower centres: "The continuum extending from deep sleep at the low activation end to 'excited' states at the high activation end is a function of the amount of cortical bombardment by the A.R.A.S.* such that the greater the cortical bombardment, the higher the activation." He also points out that there is a relationship between the level of "activation" and the level of performance (for a given performance or function) such that as activation increases performance rises towards an optimum, but, however, beyond this optimum, increase in "activation" is reflected by a fall in performance

* The ascending reticular activating system (*vide infra*).

(Fig. 1). In view of this non-linear relationship between performance and activation it will be of interest to consider other manifestations of "activation", viz. electroencephalographic activity.

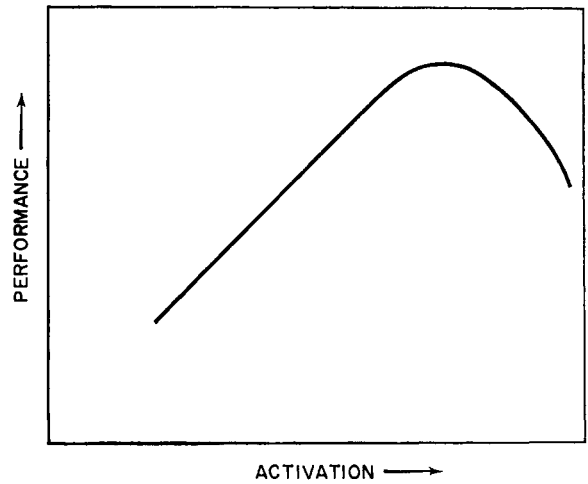


FIG. 1. A diagrammatic representation of the relationship between "Activation" and performance. For a given function, an increase in the level of activation causes a change in performance related to that function. This rises to an optimum, but thereafter declines with further increase in level of activation.

It has long been recognized that the propagation of a neural impulse is accompanied by an electrical disturbance in the region of the impulse, but it was in 1929 that Berger described phenomena fundamental to electroencephalography. Since this time, the technique of recording electrical potentials occurring at the scalp of the subject has been gradually adopted for the purpose of drawing inferences about the functioning of brain structures, and is now a commonplace clinical technique. The electroencephalogram (E.E.G.), however, does not give, because of the depth of intervening tissues, a completely accurate record of the electrical activity occuring at the surface of the cerebral cortex immediately subjacent to the electrodes. The E.E.G. is a compound of potentials derived from a variety of sources, including (1) "spike" potentials arising from discharges of cortical neurones, (2) slower changes of potential derived from local changes of potential on the dendrites of cortical neurones, and (3) potentials conducted from sources of electromotive force at some distance from the electrodes (Eccles, 1953; Marshall and Walker, 1950). Although it can be demonstrated that a mass of nervous tissue tends to have a spontaneous rhythmic discharge of its own, the rhythmic potentials developed in the cerebral cortex are influenced to a considerable degree by neural impulses arriving from subcortical structures. The "recruiting response" *(vide infra)* exemplifies this. Whilst the exact mechanism for the production of those

potentials recorded by the E.E.G. remains a matter of controversy (Walsh, 1957), certain relationships between E.E.G. records and behavioural expressions of neural activity have been noted.

The normal electrical activity recorded by the E.E.G. occurs at a frequency of 8–13 cycles per second and is known as *alpha* rhythm. This pattern of electrical activity is by no means regular. There are large inter-individual differences, and in any one individual it tends to vary irregularly both in frequency and amplitude, from moment to moment, and is capable of being analysed into a number of different frequencies. The *alpha* rhythm is most prominent in the normal individual resting with eyes closed, but tends to be "blocked" (i.e. desynchronized or replaced by more irregular wave forms of lower amplitude and higher frequency) when the eyes are open and subjected to visual stimulation (Redlich, Callahan and Mendelson, 1946). Visual stimulation is by no means alone in its ability to block the *alpha* rhythm, for any intense or unexpected stimulus or stress tends to do likewise. (These phenomena will be considered more fully below in terms of "arousal"). If, on the other hand, the subject is allowed to go to sleep, the resting *alpha* rhythm undergoes certain changes as the subject becomes drowsy and is replaced when sleep supervenes by a typical "sleep pattern." This pattern differs from the *alpha* rhythm in being of lower frequency, higher amplitude and tending to display more regularity or synchrony (Walsh, 1957; Daly and Yoss, 1957). Thus, for three different conditions, sleep, resting consciousness and consciousness with some degree of stimulation and activity, the E.E.G. tends to be found in three typical patterns in the normal subject.

Whilst this relationship between the E.E.G. and level of conscious activity holds promise of using the E.E.G. as an index of "activation", certain reservations must be borne in mind. For example, the blocking of the *alpha* rhythm by visual stimulation is by no means a universal phenomenon, for there are some people in whom the *alpha* rhythm is persistent when the eyes are open and others who are never seen to show a clearly defined *alpha* rhythm (Walsh, 1957). Again, although in many forms of unconsciousness (including those caused by central nervous system depressants and certain brain stem lesions)* a pattern similar to the 'sleep pattern' is seen in the E.E.G., in other forms of unconsciousness e.g. carotid sinus syncope and certain head injuries, the 'sleep pattern' is not produced. Also it may be noted that a pattern of large slow waves, resembling the 'sleep pattern' may be evoked by certain drugs (e.g. atropine) without any gross disturbance of consciousness (Feldberg, 1959). Similarly desynchronization of the *alpha* rhythm can be brought about by other drugs (e.g. Prostigmin) without any behavioural evidence of an increase in level of activation (Elkes, 1958). However, despite these anomalies, the high voltage slow wave pattern is considered to be a useful sign of loss of con-

* Massive lesions of the brain stem reticular formation affecting the reticular activating system (*vide infra*) result in permanent loss of consciousness. However, it has been shown that the nervous system is capable of adapting to the loss of these structures, to the extent that such lesions, if made in multiple stages instead of one stage, did not result in permanent unconsciousness (Adametz, 1959).

sciousness, and the desynchronization of the *alpha* rhythm to be a sign of increase in "cortical activation" (Feldberg, 1959).

As has been noted, the *alpha* rhythm can be "blocked" or desynchronized by an appropriate external stimulus, but this can also be brought about by internal stimuli (a non-physiological example of this being direct electrical stimulation of specific portions of the central nervous system). The behavioural manifestations that accompany these changes in the E.E.G. have a resemblance whether the stimulus producing them is external or internal. If the desynchronization is evoked by an external stimulus, it is accompanied by behavioural changes which can be interpreted by an observer as a change in the awareness of the subject so that his attention is specifically directed to the region of the stimulus and concurrent with this there are general somatic changes which can be interpreted as an increase in the alertness of the subject. If, on the other hand, the desynchronization of the E.E.G. is brought about by electrical stimulation of certain brain stem structures (the stimulus itself being not directly perceived by the subject) a similar behavioural alerting and an increase in "vigilance" takes place (Mounier and Tissot, 1958; Dell, P.C., 1958). This increase in cortical activation (in terms of E.E.G. desynchronization) or "alerting response" is also known as an "arousal response" due to the ability of both external stimulation and appropriate electrical stimulation of the brain stem to awaken or arouse a somnolent animal. The alerting response is considered to be an accompaniment of Pavlov's "orientation reflex" (Rozhe and Voronin, 1958).

The arousal produced by an external stimulus tends to vary under the influence of diverse factors. One of these is the nature of the stimulus itself – e.g. strong stimuli, in general, have more arousal value than weak ones, (nocioceptive stimuli tend to be most potent in this respect), and also a stimulus perceived through one modality of sensation may have more arousal value, by reason of the modality through which it is received, than a comparable stimulus received through another modality of sensation (Bernhaut, Gellhorn and Rasmussen, 1953). Another factor determining the strength of arousal response to stimulation is the internal state of the organism itself at the time of presentation of the stimulus – e.g. if the subject has been habituated *(vide infra)* to the stimulus prior to its presentation, arousal value is diminished, or again, if at the time of presentation the subject is motivated by the demands of one or other biological needs, the response can vary with the condition of the biological drive state (Dell, 1958). The arousal elicited by direct stimulation of certain brain structures appears to be more consistent and more intense than that in response to external stimulation (Mounier and Tissot, 1958).

Considerable knowledge has been gained over the last decade or so about the neurophysiology of these structures that are involved in the arousal response. Most investigations of this kind involve the use of techniques not applicable to human subjects and have made use of animal subjects. However, provided that intergeneric differences are kept in mind the general principles derived from animal experiments can be of great use in the interpretation of human phenomena.

The Cortex and the Reticular Formation of the Brain

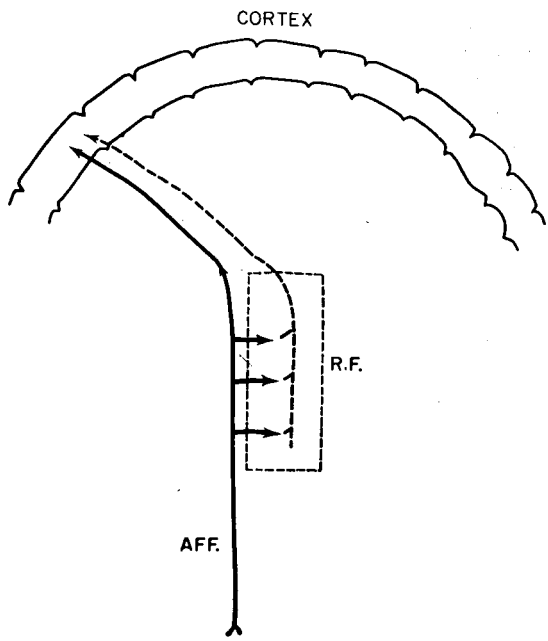
Before considering those mechanisms involved in the phenomenon of "cortical activation", it is necessary to consider briefly some aspects of the function of the neocortex in the integrative role of the central nevous system. This cortex, containing as it does the terminations of the long afferent (sensory) pathways and the origins of the great motor efferents, has been considered the highest "level" of nervous system functioning. Cortical ablation experiments have shown, however, that the functional integrity of the interrelationship between neocortex and the rest of the nervous system is not essential for a wide range of basic behavioural responses. Reflex activity (dependent on subcortical connections) remains intact, as indeed does a range of fairly complex subcortically mediated activity, e.g. crude fear and rage reactions, forms of sexual behaviour, defaecation, deglutition, chewing, walking and other fairly complex motor patterns (Bard and Macht, 1958). Plasticity in terms of the acquisition of crude conditioned responses can also be demonstrated (Walsh, 1957). The decorticate animal may also display phases of restlessness and responsiveness alternating with phases of unresponsiveness and inactivity, recognizable as representing the waking and sleeping states of the intact animal (Bard and Macht, 1958). The "sleeping" decorticate animal can be aroused by external stimulation as happens in the intact animal. Despite these findings, it is apparent that the functional interrelationship between cortex and the rest of the nervous system is necessary for memory, learning and fine discriminative behaviour (in terms of both perception and motor response) as manifest in the intact animal (Feldberg, 1959).

The nervous structures responsible for the phasic waxing and waning of overall activity in the decorticate animal and wakefulness and sleep *(vide infra)* in the intact animal lie in the reticular formation of the brain (Feldberg, 1959). These structures extend from (and are continuous with) the reticular formation of the spinal cord, up through the brain stem into the diencephalon. Roughly speaking, the term reticular formation covers that diffuse network of short aroused neurones which lie between the more anatomically discrete structures, viz. the long fibre tracts and the more discrete nuclear agglomorations, mostly serving as "relay stations" in connection with the long fibre tracts (Brodal, 1957).

The reticular formation of the brain can be considered as a pathway for conduction of impulses, accessory to the classical long afferent and efferent pathways. Some of the pathways that lie within the reticular formation have been clearly defined anatomically (Nauta and Kuppers, 1958). Peripheral stimulation, for example, causes impulses to be sent by the direct route along the long sensory tracts to the appropriate cortical projection area, whilst also, impulses are sent via collaterals from the long sensory tracts into the brain stem reticular formation through which they are transmitted and eventually also projected to the cortex (Fig. 2a) (Nauta and Kuppers, 1958; Scheibel and Scheibel, 1958). The arrival of such impulses at the sensory cortex can be recorded electrically and the primary complex of this electrical disturbance can be divided into

various phases related to the arrival of impulses via the long afferent pathways and then to following impulses arriving via the reticular formation (Brazier, 1958). The delay incurred by transmission through the reticular formation is understandable in terms of the type of neurone involved and the multisynaptic path that must be followed. Impulses arriving at the cortex via the reticular formation are not necessarily confined to the specific sensory projection area as are those traversing the terminations of the long afferent tracts, for if the stimulus is of a nature to give rise to a generalized alerting response, widespread projection of impulses from the reticular formation is evoked when the latter is subjected to bombardment via the sensory collaterals (Fig. 2b). Just as ascending impulses are routed via the reticular formation as well as along the classical pathways, descending impulses originating from the cortex are conveyed through the reticular formation as well as along the classical efferents.

Whilst impulses transmitted via the long classical pathways appear, in the main, to be concerned with the carriage of detailed sensory and executive information, those impulses travelling through and elaborated within the reticular formation appear to be concerned with facilitatory or suppressor effects capable of modulating the transmission of impulses through other centres including reflex centres, the "relay stations" related to the long classical pathways and the cortical integrating mechanisms. These facilitatory and suppressor influences may be of a general tonic kind or of a more specific, discrete and phasic nature. They will be considered in more detail below.



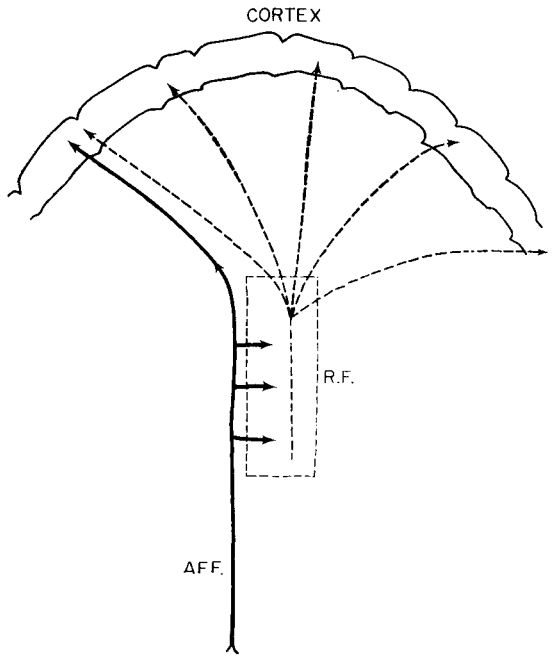


FIG. 2b. This diagram illustrates the function of the reticular formation (R.F.) as an alternative pathway for impulses proceeding from the periphery to the cortex. Impulses travelling to the cortex via the classical afferent pathways (AFF) also enter the reticular formation via collaterals from the afferents giving rise to impulses that are not only directed to the primary cortical projection area of the classical afferent (Fig. 2a), but may also be projected diffusely over a wide area of the cerebral cortex (Fig. 2b).

The peripheral influences of the reticular formation of the brain may be reviewed in relation to the following:

Sensory Input

One of the most interesting aspects of reticular formation functioning is its involvement in the "gating" of incoming sensory information. Such information is not just passively conveyed from the periphery to the cortex but a process of selection takes place in terms of a facilitation or a suppressor action on the functioning of the peripheral "relays" involved in the transmission of such information via the classical afferents.

Briefly some of the findings which indicate the existence of this mechanism are: By Granit (1955) — The retinal relays are usually enhanced but may be inhibited by reticular formation stimulation. Hernandez Peon, Guzman—Flores, Alcaraz and Fernandez—Guardiola (1958) suggest that the process of habituation to repeated visual stimulation takes place at least at the level of the lateral geniculate body through centrifugal inhibition of reticular origin. Rasmussen (1946; 1953), and Galambos (1954, 1956) have shown similarly suppression of auditory nerve impulses in response to reticular formation stimulation, whilst Hernandez Peon, Lavin, Alcocer—Cuaron and Marcelin (1960) have studied centrally determined modification of the activity of the olfactory bulb.

Motor Outflow

In the same manner as there is a control of incoming impulses, there is also a reticular formation influence on effector impulses, which depends on the facilitation or suppression of the functioning of the structures conveying them.

The Corticopetal Influences of the Reticular Formation

Cortical "Facilitation"

As has been mentioned previously, desynchronization of the *alpha* rhythm accompanied by behavioural arousal can be evoked by stimulation of portions of the reticular formation either by direct implantation of electrodes or by peripheral sensory stimulation (via the collaterals of the afferents). This phenomenon is thought to be due to the bombardment of the neocortex by facilitatory impulses derived from the reticular formation and transmitted through that part of the reticular formation known as the ascending reticular activating system (Walsh, 1957). This cortical facilitation may be specifically localized to areas of the cortex or it may be a non-specific widespread phenomenon.

Suppressor Influences

It is evident from numerous studies *(vide infra)* that an active kind of inhibitory influence can be exerted by portions of the reticular formation. That portion of the reticular formation known as the "recruiting system" exemplifies this property. The recruiting system when stimulated electrically in a particular way gives rise to an increase in amplitude and a synchronization of cortical potentials. The relationship of this imposed synchrony to that which occurs in states of low activation has been demonstrated by Hess (1954) who has shown that stimulation of the intraluminar nuclei of the thalamus (in cats) by low frequency electrical stimulation gave rise to a synchronized rhythm resembling the sleep pattern of the E.E.G. and accompanied by behaviour changes similar to those occurring in somnolence, which outlasted the duration of the stimulation. Thus, it can be seen that the reticular formation exerts both suppressor and facilitatory influences of both a non-specific, tonic kind and also a localized specific kind both peripherally and cortically. Moreover, it is thought that the influence of the reticular formation is not confined to cortex and periphery alone, but occurs at all levels of the central nervous system (Jasper, 1958).

Some Aspects of Brain Functioning and Behaviour

It is now possible to conceive of the reticular formation as being a dual structure containing elements with a "facilitatory" function (the activating system) and others with an "inhibitory" function (synchronizing elements).

The Activating System

The phenomenon of electrophysiological activation (E.E.G. desynchronization accompanied by behavioural arousal) can be obtained by stimulation of wide areas of the lateral portions of the reticular formation of the medulla, pons and midbrain and also throughout areas of the diencephalic reticular formation. Of these areas note may be taken of several of the most important:

(1) a region in the rostral portion of the pons appears to be of critical importance in the maintenance of wakefulness.

If this area is destroyed, the activating influence of the midbrain reticular formation and other areas are not by themselves sufficient to maintain normal alert wakefulness. If, however, the suppressor elements caudal to this region are destroyed, a state of continued vigilance supervenes (Moruzzi, 1960).

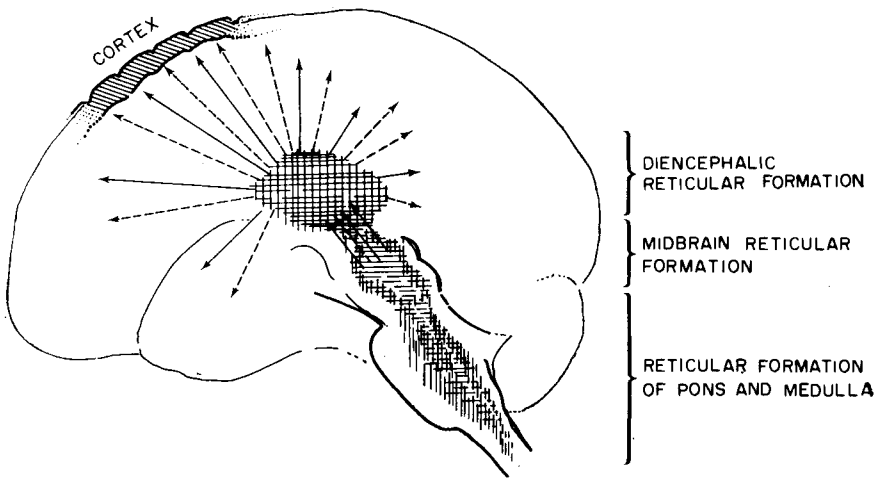


FIG. 3. A diagrammatic representation of the reticular formation showing the brain stem reticular formation which gives rise to facilitatory impulses of a nonspecific kind arising from areas in medulla, pons and midbrain (the latter containing adrenaline sensitive elements) which are directed up through the diencephalic reticular formation. (These activating impulses arising from the brain stem are of tonic kind, whilst impulses arising due to stimulation of the thalamic reticular formation within the diencephalon give rise to a phasic activation.) Also lying in the reticular formation are synchronizing elements (of note, within the medulla, pons and recruiting system of the thalamic reticular formation) which give rise to impulses directed at the cortex antagonistic in effect to the influence of the activating system.

(2) The midbrain activating system is of note in that it is sensitive to and stimulated by adrenaline and allied substances (Dell, 1958).

(3) The thalamic portion is of note in that it contrasts with the brain stem activating system in the following particulars. The thalamic portion

projects to the cortex so that stimulation of a discrete portion of this system tends to give rise to a specific localized cortical activation; moreover, it is thought that the thalamic activating system mediates the rapid, short lasting or phasic activation of the cortex in contrast with the slower, longer lasting tonic activation dependent on the brain stem activating system. It is also believed that the activation evoked by stimulation of the tonic portion of the activating system is easily habituated whereas that evoked from the phasic portion is habituated only with difficulty and recovers more rapidly (Jasper, 1958) (Fig. 3).

Suppressor Mechanisms

In the diencephalic portion of the reticular formation, the recruiting system is thought to be functionally antagonistic to the diencephalic activating system (with which it coexists in this region) (Tissot and Mounier, 1959). The suppressor mechanisms (synchronizing elements) that have been described as existing in the brain stem, also appear to be functionally antagonistic to the activating system (e.g. if the suppressor mechanisms in the caudal portion of the brain stem are blocked by local injection of barbiturate, generalized activation is the result (Moruzzi, 1960) (Fig. 3).

So far we have considered mainly stimulation of the reticular activating system in terms of either direct electrical stimulation or impulses that reach it via the collaterals of the afferents. The reticular formation as a whole, however, is influenced directly or indirectly by neural impulses arising in diverse portions of the nervous system and also by humoral "messages" relating to the state of the "*milieu interieur.*" Moreover, the interrelationship between the reticular formation and the rest of the central nervous system is more complex than has been herein indicated so far.

A brief consideration of some of the more complex interactions between reticular formation and the rest of the central nervous system having some relationship to behavioural phenomena is:

(I) *Neural "feed-back" mechanisms*

(a) *Cortex and reticular formation* — In the foregoing pages, stress has been laid on the influence of the reticular formation on the level of cortical activity. However, the mechanism exists for the cortex to influence the functioning of the reticular formation. Both alerting and recruiting responses can be obtained by direct electrical stimulation of appropriate cortical areas (Walsh, 1957; French, 1958).

(b) *Peripheral structures and reticular formation* — As has been noted, the reticular formation besides influencing both the functioning of effector organs and sensory input, is itself influenced by sensory input derived both from exteroceptors and interoceptors related to effector organs. For example, generalized activation tends to lead to an increase in skeletal muscle tone; proprioceptive impulses derived from increased levels of skeletal muscle tone have of themselves an activating value (Livingstone, 1958; Ward, 1958) and tend to maintain activation. The lowering of the level of activation which accompanies muscular relaxation may be associated with an interference with positive feed-back mechanisms (Moruzzi, 1960).

(c) *The vegetative nervous system and the reticular formation* — Fluctuations of the level of activation as indicated by E.E.G. recording have been observed to be associated with fluctuation in general autonomic tone (Bonvallet, Dell and Hiebel, 1954). Activation tends to be accompanied by the release of adrenal hormones which in turn have an action on the adrenaline sensitive portions of the midbrain activating system tending to potentiate the activation. On the other hand, a rise in blood pressure (such as may be found

with release of adrenaline, in physical exercise, in states of emotion etc.) stimulates carotid sinus pressor receptors which by their central nervous connections tend to have a damping effect on the level of activation (Dell, 1958)

(2) *Alterations in the milieu interieur.*

The reticular formation plays a role in the generalized increase in the level of activity which is associated with certain biological "drive states." For example, food deprivation tends to be followed by a reduction in the normal resting blood sugar value and a compensating release of adrenaline which tends to restore the blood sugar. This increase in the level of circulating adrenaline provides a stimulus to the adrenaline sensitive midbrain activating system giving rise thereby to a tonic generalized activation. Another example found in the lowering of threshold for activation (evoked by direct electrical stimulation) that occurs with artificial oestrous induced by the appropriate administration of homenes (Dell, 1958).

(3) *Vigilance, attention, perception, habituation and conditioning*

Electrophysiological activation is conceived as being accompanied by an increase in alertness and the function of vigilance which is reflected by alteration in performance involving perceptual abilities (bearing in mind the non-linear relationship between performance level of activation *(vide supra* and Fig. 1)). It has been shown by Fuster (1958) that monkeys show improved perceptual discrimination when their reticular activating system is subject to stimulation. Perceptual discrimination would seem to involve the ability to attend to relevant sensory data to the exclusion of irrelevant sensory data. That the reticular formation plays a part in the suppression of irrelevant sensory signals (at a peripheral level) when attention is focused in another field of perception has been demonstrated by Hernandez Peon, Scherrer and Jouvet (1956): a cat resting quiet surroundings displays quite sizable potentials recorded from the auditory nerve in response to auditory stimulation; if a mouse is introduced so that the animal's attention is diverted by its appearance, the response to stimulations at the auditory nerve is damped down appreciably suppressor impulses from the reticular formation, but return again to their previous magnitude when the mouse is removed and the cat settles down. Another aspect of the problem of perceptual discrimination and attention has been demonstrated by Kogan (1960) who has shown that cortical desynchronization during a sensory stimulus was accompanied by a decrease in threshold in the corresponding primary receiving area, along with an increase in threshold of other cortical areas.

The phenomenon of habituation, discussed previously in relation to the peripheral suppressor effects of the reticular formation, would seem to be a mechanism whereby a contrast in value between familiar stimuli and novel stimuli was attained, so that attention tends to be directed towards novel stimuli in preference to familiar stimuli. The specificty of habituation, for example, in terms of frequency when the stimulus is a flickering light has been demonstrated by John and Killam (1959). The importance of the role played by the reticular formation on habituation is indicated by the "dishabituation" (return of a previously habituated response) that can be brought about by lesions of the reticular formation or the administration of pentobarbitone sodium (John, 1961).

Habituation seems also to play a part in the conditioning situation in that an indifferent stimulus repeatedly coupled with an alerting stimulus does not habituate, as does an indifferent stimulus on its own (Gastaut, 1958). Jasper (1958) believes that the habituation of irrelevant stimuli in conditioning is a significant factor in forming a conditioned response involving the experimental stimuli.

With regard to conditioning itself, whilst a contrast between significant and irrelevant stimuli may involve the habituation of irrelevant stimuli, another process is in operation at the same time: the previously indifferent stimulus gains in its ability to produce cortical "excitarion" in terms of its ability to provoke desynchronization of the E.E.G. For example, a stimulus previously incapable of giving rise to desynchronization does so when it has been coupled many times with an alerting stimulus viz. proprioceptive and other impulses derived from an active or passive displacement of the hand (Gastaut, 1959). Gastaut's views about the mechanisms involved in, and the events occuring with conditioning are:

(a) *Site of formation of temporary "connection"* — This is believed to occur in the reticular formation. The formation of an initial cortico-cortical "connection" lying within the cortex itself seems unlikely in that conditioning can take place even when the cortical

analysers related to the conditioned and unconditioned stimuli are separated by an incision in the cortex, or even when the cortical analyser related to the *conditioned stimulus* is ablated. This view seems also to be supported by the electrical events which accompany the formation of a conditioned response.

(b) *The electrical activity of the cortex during conditioning* — Using an unconditioned stimulus of an intensity that evokes a desynchronization localized to the area of its cortical analyser, and an indifferent stimulus (i. e. the stimulus that is to be conditioned) which initially does not evoke E.E.G. activation, it is found that as conditioning takes place the conditioned stimulus (preciously indifferent) acquires the ability to alter the E. E. G. in the same way as the unconditioned stimulus, i.e it gives rise to a desynchronization localized to the area of the cortical analyser of the *unconditioned stimulus*, and is believed to have acquired the ability to activate that portion of the reticular activating system which projects to the cortical analyser of the *unconditioned stimulus*. This acquisition of some of the electrophysiological properties of the unconditioned stimulus by the conditioned stimulus is also accompanied by the acquisition of some of the former's behavioural properties in terms of response

(c) *Electrical events related to the inhibition of a conditioned response* — (i) During the development of *conditioned inhibition* (e. g. extinction and differentiation) there is observed an increase in the amplitude (i. e. a tendency to synchrony) of the spontaneous rhythm localized to the area of the unconditioned cortical analyser which was previously the site of localized desynchronization. (ii) During *external inhibition* (accompanying the use of a disturbing stimulus) a generalized desynchronization ("activation") is observed. (This is of interest in terms of the general finding of non-linear relationship between activation and performance *(vide supra* and Fig. 1).) (iii) During *supra maximal inhibition* (accompanying a repetive stimulus) there is first a generalized amplification of cortical rhythms followed by slowing and transition into sleep rhythms.

Sleep

It has been noted that as sleep supervenes the resting *alpha* rhythm is replaced by the so-called "sleep" rhythm in the E.E.G. Besides the superficially observable postural changes, there is a lowering in the level of general body functioning (indicated by such things, for example, as lowering of skeletal muscle tone, a fall in the level of circulating adrenaline, a slowing of pulse and respiration etc.) as sleep deepens. Along with these phenomena goes an apparent lowering of responsiveness to external stimuli and other evidence of a diminution of general nervous activity (e.g. an accumulation of energy rich substances in the cerebral hemispheres, which are normally utilized during wakefulness (McIlwain, 1959). This apparent "shut-down" of central nervous activity and the changes in the tone of bodily functioning that accompany it, has been ascribed to alterations in the level of functional activity of various portions of the reticular formation.

Moruzzi (1960) favours the opinion of those who hold there are two types of sleep, sleep as a passive phenomenon and sleep as an active phenomenon. Passive sleep can be thought of as resulting from a withdrawal of the activating influences stemming from the reticular formation. This process becomes progressive and is maintained by the diminution of those influences feeding back onto the reticular activating system which help maintain its tone during wakefulness (e.g. diminution of corticofugal feedback, proprioceptive inflow, level of circulating adrenaline etc.). An example of this kind of passive sleep is that which supervenes under conditions of sensory deprivation with the reduction of the activating influence of tonic sensory inflow.

Sleep as an active phenomenon is exemplified by that which supervenes in relationship to repetitive sensory stimulation, e.g. as accruing from "supramaximal" inhibition (Gastaut, 1958) or subsequent to the extinction of a conditioned reflex. It has been postulated that the habituation occurring with repetitive stimulation (representing in effect a de-afferentation taking place both peripherally and centrally) results in a diminution in the activating influence of sensory inflow and leads to a situation comparable to sensory deprivation. However, Moruzzi believes this kind of explanation inadequate for a variety of reasons (e.g. it cannot explain the rapidity of onset of sleep in some instances). He suggests that repetitive stimulation of this kind actively stimulates the synchronizing elements of the reticular formation and leads to a depression of the activating system and a lowering general nervous activity. During conditioning, the activating influence of the unconditioned stimulus would tend to antagonize the synchronizing effects of the repetition of the indifferent stimulus. During extinction, the activating influence (acquired from the unconditioned stimulus) of the now conditioned stimulus would also serve to maintain the level of activation. However, after extinction, when the indifferent stimulus no longer evokes activation, the effect of its repetition upon the synchronizing elements would be unmasked leading to a widespread inhibition.

General Central Nervous System Depressant Drugs

Drugs having a depressant effect on the functions of the central nervous system may be classified into those having a more general effect (general anaesthetics, hypnotics and sedatives) and those having specific depressant properties (e.g. anti-epileptics, narcotics, and ataractics). It is the former group that will be considered at present.

The apparent differences between the predominant action of the general anaesthetics and the hypnotics and sedative group are more a function of their usage than differences in basic biological activity. The anaesthetics are substances which lend themselves to modes of administration (by inhalation or intravenous injection) allowing a rapid attainment of pharmacologically active blood levels, and moreover, have the property of being subject to a rapid fall in activity (by reason of rapid elimination through the lungs, rapid destruction through metabolic processes, or rapid dilution by diffusion from the blood stream into the general body fluids) as soon as administration ceases. Sedatives and hypnotics, on the other hand, are substances more suited to oral or intramuscular administration, which, in general, also require a period of some hours for de-activation and elimination by the body. In speaking of the general anaesthetics, McIlwain (1959) states: "The loss of sensation caused by these substances – ether, chloroform, nitrous oxide or cyclopropane – is accompanied by loss of consciousness, and their effects, considered collectively, are not clearly distinguishable except in degree from those of the class of depressants more frequently used as hypnotics, for example, the barbiturates."

In trying to determine the essential features common to this group of substances (differing widely in chemical structure and physical properties) which is related to their action on the central nervous system, it is interesting to consider some other features common to the group as a whole. Firstly, they have in common other biological properties — e.g. inhibition of cell division, photosynthesis, bioluminescence — and the degree of activity displayed in one aspect of their biological activity is usually related to the degree of activity displayed in others. This relationship tends to be less precise for the more chemically active substances, than for those which are less chemically active (nitrogen, nitrous oxide) or chemically inert (krypton, xenon). Secondly, it is possible to correlate the degree of biological activity they display with some of their purely physical properties, e.g. lipid solubility, vapour pressure, liquid–liquid distribution, which are in turn related to their thermodynamic activity (McIlwain, 1959). From this it would seem that the thermodynamic activity of these substances was perhaps of more importance in regard to their biological activity than their actual chemical properties, though the latter would be relevant in that the more chemically active substances would tend to undergo rapid chemical changes within the body.

The site of action of general depressant drugs within the nervous system is believed to be upon those structures concerned with the synaptic transmission of neural impulses rather than upon axonal conduction* (McIlwain, 1959). If this is generally true, it would be expected that the transmission of neural impulses through polysynaptic structures would be interfered with to a much greater extent than transmission through long fibre pathways involving few synapses – this appears to be the case when we consider the ability of anaesthesia to block transmission through the reticular formation (a polysynaptic structure) whilst leaving intact transmission through the great afferent and efferent pathways which contain few synapses (Killam and Killam, 1958; Brazier, 1958; Feldberg, 1959). Thus electrophysiological activation can be blocked by an anaesthetic agent without preventing sensory impulses from reaching the cortex; the sensory impulses apparently losing significance when unaccompanied by an appropriate level of cortical activation. The effects of these depressant drugs upon the central nervous system have generally been attributed to a selective interference with the reticular activating system, so that a state of lowered cortical activation ensues (Moruzzi and Magoun, 1949; Arduini and Arduini, 1954). Moruzzi (1960), however, now believes that at least for very low levels of anaesthesia, the co-operation of a synchronizing influence arising from the lower brain stem is of considerable importance in producing a lowering of a level of cortical activation, rather than the latter being the result of an exclusively selective action on the activating system.

With the administration of an anaesthetic or a large dose of sedative drug, events tend to occur in a sequence involving progressive modification of consciousness and bodily functions. Four stages of anaesthesia are usually recognized clinically (Lee, 1959).

* Ethyl alcohol may be atypical in this regard (Larrabee & Pasternak, 1952).

(1)*A phase of induction* in which, although mental control and consciousness are present, these are modified by the marked hypnotic effect of the anaesthetic and are accompanied by a progressive analgesia (but not total abolition of pain sensation). This phase passes into the subsequent one with loss of conscious control.

(2) *A stage of "excitement"* (delirium) in which there may be struggling, breath-holding, vomiting or coughing. Reflexes are usually present and exaggerated. There are marked individual differences in the occurrence and duration of this stage.

(3) *The stage of surgical anaesthesia*, characterized by unconsciousness, absence of gross response to painful stimuli, "automatic" breathing, progressive muscular depression and depression of reflex functions. The latter tend to disappear in a regular order as the depth of the anaesthesia is increased.

(4) *Stage of respiratory paralysis*, which is in turn followed by circulatory failure and death.

With a large dose of sedative or hypnotic drug the following stages can be recognised (Wilson and Schild, 1959).

(1) *Lessened movement or Hypnosis.* There appears to be a slowing of thought processes. The coordination of the limbs is impaired. Normal sleep from which the subject is easily aroused tends to occur. Response to painful stimuli is not interfered with to any marked extent.

(2) *Reflex depression.* Consciousness and the sensation of pain are both present but coordination is impaired and reflexes are sluggish.

(3) *Inebriation.* The subject responds to stimuli but few signs of conscious control are present. There may be restlessness which corresponds to the state of "excitement" seen with volatile anaesthetics. Pain sensation is only slightly depressed. Memory is impaired, so that the occurrence of pain is forgotten as soon as it ceases.

(4) *Anaesthesia* occurs as with the inhalation anaesthetics. Respiratory failure, circulatory failure and death can follow.

Clinically, the chief difference between anaesthetics and the hypnotics and sedatives is that, with the latter there is no marked phase of excitement. This "excitement" is a variable phenomenon varying not only from drug, and from subject to subject, but also within one subject, from occasion to occasion. It is likely to be more pronounced with those anaesthetics which are irritant to the respiratory tract and is supposed to occur more readily in those people who are heavy smokers and drinkers. The phenomenon has been ascribed to a possible direct action of drug on cortex (Brazier, 1953), but it must be borne in mind that the reticular formation as a whole (both synchronizing elements and activating system) appears to be susceptible to general depressant drugs (as instanced by the alerting response obtainable with pentothal injected so as to effect the mainly synchronizing elements of the lower brain stem (Moruzzi, 1960)). Whatever the exact mechanism of the production of this phase of excitement, Wilson and Schild (1959) are of the opinion that it is related to the circumstances and surroundings in which the drug is administered.

The anaesthetic precedes traumatic surgical intervention, with the latter's attendant emotional disturbance, the hypnotic, however, is taken in the quiet of retiring to bed in the anticipation of sleep. The importance of protecting the individual who has taken a sedative or hypnotic drug from excessive external stimulation, lest an inebriated state ensue is well known.

The action of the general depressant drugs, in lower levels of dosage, can be thought of in several ways. One can place the emphasis on their action on the reticular formation, and regard the effects that follow administration as a manifestation of a shift in the balance between the ac-

tivating and the synchronizing elements which, in turn, affects both cortex and more peripheral nervous structures. On the other hand, this conception could be restated in a more general fashion by considering cortex reticular formation and allied structures, as a functionally integrated unit, and that the effect of the drug upon this unit was to bring about a shift in the balance of "excitation"–"inhibition"* levels within this unit. Much work has been done on the effects of general central nervous system depressants on behaviour. An excellent review of this kind of work may be obtained by referring to Trouton and Eysenck (1960). A brief appraisal of some general depressant drugs is shown below (Table 1) for the purposes of comparison.

TABLE 1

Nitrous Oxide	Inorganic gas	A weak inhalation anaesthetic usually supplemented by other depressant drugs.
Ethylene (C_2H_4)	Unsaturated aliphatic gas	Inhalation anaesthetic somewhat more potent than nitrous oxide.
Cyclopropane (Trimethylene) ($CH_2)_3$	Cyclic hydrocarbon gas	A potent and potentially toxic inhalation anaesthetic.
Ether (Ethyl Ether)	Volatile liquid	Inhalation anaesthetic.
Chloroform	Volatile liquid	A potent and potentially toxic inhalation anaesthetic.

* The words "excitation" and "inhibition" as applied by the psychologist are hypothetical constructs and must not be confused with the same words as used in a technical sense by the neurophysiologist. The neurophysiologist uses "excitability" to describe a definite property of a single neurone. The word "excitation" is used by the physiologist in a related sense when speaking of the influence of a stimulus on single neurone or small group of neurones. The word "inhibition" is used to describe the lowering of the level of excitability that occurs in a single neurone or small group of neurones under certain circumstances (e.g. the inhibitory influence of the Renshaw cell on the spinal cord motor neurone). However when describing the functional activity of a large number of neurones composed of both neurones having an "inhibitory" influence and an "excitatory" influence interacting in a complex fashion, the physiological terms "excitation" and "inhibition" as applied to units become meaningless, so that when speackng about molar activity the physiologist tends to apply the words "facilitation" and "suppression" in regard to the gross influence of one part of the nervous system or another.

TABLE 1 (continued)

Thiopentone Sodium (Pentothal)	A water soluble solid barbituric acid derivative	Intravenous anaesthetic. Very short acting in comparison to other barbiturates.
Quinalbarbitone (seconal sodium)	A barbituric acid derivative	Oral hypnotic. Length of action 2–3 hours.
Amylobarbitone Sodium (Amytal sodium)	Barbituric acid derivative	Oral hypnotic of 6–8 hours duration. Also used as a sedative given intramuscularly or intravenously.
Phenobarbitone (Luminal)	Barbituric acid derivative	Oral hypnotic of 8–12 hours length of action. Also used as an intramuscular sedative.
Paraldehyde	Water soluble oily liquid	Oral hypnotic. Sometimes used as a basal anaesthetic.
Glutethimide (Doriden)	Chemically related to phenobarbitone	Oral hypnotic – rapidly acting and lasting about 6 hours.
Methyl Pentynol (Oblivon)	An unsaturated higher alcohol	An orally administered mild sedative used as a "tranquillizer".

Atypical and Specific Central Nervous System Depressants

(1) *Ethyl Alcohol.* Many of the actions of these substances are similar to those of the general central nervous system depressants, but it produces a more pronounced "inebriation." It appears to have a greater tendency to suppress axonal conduction as well as synaptic conduction than do most of the general depressants (McIlwain, 1959).

(2) *The Analgesics and Antipyretics*, e.g. Aspirin (acetyl salicylic acid). Whilst some of these substances tend to have a mild sedative action, their

main actions are in terms of their effect on body temperature in febrile states and pain relieving properties.

(3) *The Opium Alkaloids and derivatives.* These substances whilst having a marked central depressant action, tend to interfere with some functions much more than others, viz. pain sensibility and some centres of vegetative functioning (e.g. respiratory centre). Some have marked peripheral effects of a cholinergic kind. Addiction is a feature of their repeated usage.

(4) *The Ataractics.* These are a diverse group of drugs used for their "tranquillizing" properties. Two important examples are:

(a) *Phenothiazine derivatives* (e.g. chlorpromazine (Largactil), thorazine) – Chlorpromazine has a number of pharmacological actions. It tends to potentiate the action of hypnotics and anaesthetics, whilst it, itself, is not an anaesthetic agent. It has effects on various centres, by reason of which it demonstrates an ability to lower normal body temperature. It also has a mephenesin-like action and adrenolytic properties. Following administration the subject lies quietly and indifferently but without clouding of consciousness. Reaction time, tapping speed and memory for numbers may be impaired. It is thought to interfere with the alerting effect of impulses impinging on the reticular formation via the sensory collaterals, so that it diminishes tonic activation without affecting phasic activation (Bradley, 1958; Killam and Killam, 1958).

(b) *Meprobamate* (Miltown, Equanil) – This drug has a mild sedative action, but its predominant effects are those of a centrally acting muscle relaxant. The action of meprobamate on the spinal cord resembles that of mephanesin in that it inhibits reflexes involving several internuncial neurones, thus diminishing muscle tone. (This reduction in muscle tone would possibly tend to reduce activation dependent upon tonic proprioceptive inflow.)

(5) *The Anti-epileptics.* Anticonvulsant drugs are thought to act by preventing the spread of the abnormal discharges in the brain that are associated with epilepsy. Until fairly recently, sedative and hypnotic drugs were used for this purpose, but now a range of drugs having relatively specific anticonvulsant properties have been built up (e.g. primidone (Mysoline), phenytoin sodium (Epauntin).

Central Nervous System Stimulants

These drugs may be divided into those having a general action on central nervous system functioning and those having more specific properties (e.g. convulsants and analeptics). The latter will not be considered in detail and the former are a comparatively small group which include: amphetamine and related compounds, caffeine, pipradol (Meratran), methylphenidate (Ritalin) and cocaine.

The Amphetamines

These drugs have a close relationship to naturally occurring adrenaline. The peripheral effects of adrenaline (epinephrine) and noradrenaline (norepinephrine, levarterenol) in the functioning of the autonomic nervous system, their metabolic effects and the similarities and differences between

them are well detailed and will not be considered here. Their central effects, however, are quite relevant — they both (with approximately equivalent potency) produce electrophysiological activation when given intravenously (Rothballer, 1956). This action is thought to be mediated by a specifically adrenaline sensitive portion of the midbrain activating system and is tonic rather than phasic. (e.g. The slow delayed component of the activation that accompanies a startle response is thought to be mediated via a release of adrenaline.) It is thought that amphetamine and its relatives have an action similar to adrenaline with regard to the midbrain activating system (Bradley and Elkes, 1957), but the duration is not as evanescent and the peripheral effects not as marked as with adrenaline.

Amphetamine and similar substances besides having central nervous system stimulant properties have peripheral actions of a sympathomimetic kind. These peripheral effects vary from drug to drug. They also have in common a tendency to produce anoerexia and mood changes (usually euphoria though sometimes "irritability") but vary also from drug to drug in this respect. Their stimulant effect may be followed when the pharmacological action ceases by a depression of mood. Some of these substances when taken excessively over a period of time are liable to lead to a toxic psychosis resembling schizophrenia.

Amphetamine sulphate (Benzedrine) is the racaemic mixture of the *laevo* and *dextro* forms. The *laevo* form has more peripheral action and less central action than the *dextro* form — *dexamphetamine sulphate (Dexedrine)* which is a potent central nervous system stimulant. The latter has been used medicinally as a euphoriant, in the treatment of some types of psychopathy (Sargant and Slater, 1954), as an appetite depressant in obesity, in the treatment of narcolepsy and also in sedative overdosage. *Metylamphetamine hydrochloride (Methedrine, Pervitin)* is slightly more active as a central stimulant than dexamphetamine, but has more peripheral side effects.

Ephedrine hydrochloride has central stimulant properties similar to the above, but these are overshadowed by its peripheral effects.

Other Central Nervous System Stimulants

Pipradol (Meretran)

This produces an excitement similar to that produced by amphetamine, but it does not affect the blood pressure or heart rate as does amphetamine, nor does it have the appetite depressant effect of the latter. This drug activates the cortical and subcortical E.E.G. (in rabbits) (Himwich, 1959).

Methylphenidate (Ritalin)

This has a central stimulant action resembling that of amphetamine. It has some peripheral effects in that it causes a mild rise in blood pressure and an increase in heart rate. The drug appears to act as a diencephalic or thalamic level on the phasic portion of the reticular formation system and not on the tonic midbrain portion in contrast to amphetamine (Jouvet and Courzon, 1959).

It causes cortical activation (in cats) which is not abolished by lesions of the reticular formation at the level of the red nucleus but is abolished by lesions of the diffuse thalamic projection.

Stone (1960) believes that methylphenidate produces a stimulation leading to directed activity rather than to the generalized overstimulation produced by amphetamine in comparable doses, which tends to interfere

with performance. Jasmin and Bois (1959) have observed that with animals trained to perform a particular task, the drug improved their performance and delayed the onset of fatigue, but in untrained animals it increased their coordination but did not tend to compensate for lack of training.

These findings could be interpreted in terms of behavioural psychology as indicating an increase in "habit strength" due to the influence of the drug.

Caffeine

This substance produces a central nervous system stimulant effect. In large doses it produces excitement, delirium, hallucinations and possibly convulsions (Wilson and Schild, 1959). This drug appears to differ from both Ritalin and the amphetamines in its site and mode of action. It causes electrophysiological activation even when there are lesions of the diencephalic reticular formation, so that it possibly is capable of a direct action on the cortex itself. It is also believed to be able to suppress directly the activity of the thalamic synchronizing elements (recruiting system).

Cocaine

This drug also produces an excitement and, like caffeine, can give rise to convulsions in toxic doses. It chiefly facilitates motor response, making reactions quicker but less accurate. It can give rise to a narcotic addiction.

Atypical and Specific Central Nervous System Stimulants

The analeptics

These drugs like caffeine and cocaine are convulsants in larger doses. They are used (as are the amphetamines and caffeine) to counteract overdosage of C.N.S. depressant drugs. Some of them are used to stimulate failing respiration and circulation in physical illnesses, whilst others have been used to produce convulsions for the treatment of certain psychiatric disorders. Examples of this group of drugs are: picrotoxin; leptazol (Metrazol, Cardiazol); nikethamide (Coramine); bemegride (Megimide).

Strychnine

This is a convulsant. Although it has some action on all parts of the brain, its main site of action is on the spinal cord. (Its action is to interfere with the functioning of those inhibitory neurones which have a suppressor influence on the spinal cord motor neurones, releasing the latter so that the motor elements of the spinal cord become hyperirritable (Eccless, 1952).)

Lysergic Acid Diethylamide (L.S.D. 25)

This is an hallucinogen with stimulant properties. It produces an electrophysiological activation which is thought to be a function of incoming sensory stimuli (affecting the reticular formation via the afferent collaterals.) It does not produce a marked change in the threshold of the reticular formation to direct stimulation but does decrease the recruiting response in the cat (Baradley, 1958; Purpura, 1956).

Behavioural Effects of the Stimulants

Much work has been done in the study of the effect of stimulant drugs on behaviour, an excellent review of this kind of work may be obtained by referring to Trouton and Eysenck (1960).

With regard to the amphetamines, because of their physiological relationship to adrenaline it may not be possible to consider that the stimulant

effects of these drugs is completely divorced from an increase, expressed in behavioural terminology, of "drive." On the other hand, methylphenidate does not appear to act on the adrenaline sensitive portion of the reticular formation but shows some indication of increasing "habit strength." A comparison of the behavioural effects of these two stimulants may be rewarding in elucidating these relationships.

General Pharmacological Considerations

In considering general depressants and stimulants, it has been pointed out that whilst these drugs may be regarded as altering the level of cortical activation, there is a non-linear relationship between this level of cortical activation and behavioural performance. Now when we come to consider the relationship between drug dosage and the behavioural effect of the drug, a number of factors exist that make this relationship a rather complex one. If we simplify the question by supposing that a given concentration of drug is needed at the point at which it acts to produce a given effect on the nervous system, then we can first of all consider some of the more important factors concerned in the relationship between dosage and drug concentration at the "target organ." With this simplified scheme we need to take into account the absorption, distribution and inactivation of the drug, as variables to the build-up of pharmacologically active concentrations of drug.

Absorption

Inhalation is a common mode of administration of a gaseous or volatile drug agent. An example of this is the administration of nitrous oxide. This gas is soluble in body fluids but does not enter into chemical composition within the body. In passing from the lungs to the blood and from the blood to the tissues, it obeys the physical laws applicable to the solution and diffusion of gases in liquids (Lee, 1959). In passing from the external atmosphere to the alveoli of the lungs (from which it is absorbed into the blood) it obeys those physical laws relating to mixtures of gases and vapours. Individual variation arises in terms of rate of respiration, tidal volume, amount of air remaining in the alveoli and air passages at the end of each expiration, and the area of contact between the alveolar air and the capillary network of alveoli.

Intravenous injection is found a suitable means of administration for some water soluble substances such as Pentothal.

Oral administration is used for a wide range of liquid and solid drug agents. Some substances such as ethyl alcohol are absorbed through the gastric mucous membrane (Wilson and Schild, 1959), but most orally ingested drugs are absorbed through the small intestine. Besides variation in absorption dependent on the chemical and physical properties of the drug itself, variation in absorption arises in terms of such factors as gastric and intestinal mobility at the time of ingestion of the drug, the state of the contents of the gut and the vascularity of the wall of the stomach and intestine.

Distribution

With inhaled substances there is a lag between the beginning of administration and the reaching of a pharmacologically active level of the drug at the "target organ" in terms of (1) the rate of build of concentration in the alveolar air, and (2) the rate of carriage of the drug in the blood stream, away from the lungs via the pulmonary artery from whence it enters the heart and is distributed through the systemic circulation (dependent on the circulation time of the individual at the time of administration).

With intravenous injection, the substance is carried to the heart, round the pulmonary circulation and then back to the heart to be distributed in the systemic circulation. There is a lag in terms of circulation time. Also, the drug may be carried in the blood stream

in the form of a concentrated "lug" before it becomes more evenly distributed through the blood stream (Paton, 1960).

Substances ingested are carried in the portal blood stream and have to pass through the liver before reaching the general systemic circulation. This passage through the liver is important with those substances which may be destroyed by the liver, so that some variation in the amount of substance reaching the central nervous system may be in terms of individual hepatic efficiency.

Substances circulating in the systemic blood are not evenly distributed throughout the body in that those organs copiously supplied with blood (e.g. the brain) are more exposed to the influence of the drug than those tissues poorly supplied with blood (e.g. fat, cartilage). This can be illustrated by considering, for example, a substance such as nitrous oxide where a high concentration in the blood flowing to the brain results in the rapid build-up of the cerebral content of the anaesthetic whilst very little has yet reached the general body tissues. If in this condition the nitrous oxide is withdrawn, there is a rapid fall in the cerebral content as the blood content also falls rapidly. If, however, the same percentage of nitrous oxide is administered over several hours, the body tissues become saturated and an equilibrium is reached, so that cessation of administration results in a much slower fall in cerebral content, owing to the storage of the anaesthetic throughout the body (Willson and Schild, 1959).

Another factor relating to distribution of drug in the body is in terms of selective absorption of the drug by a particular organ or tissue. The demonstration of a "blood-brain barrier" specific to particular substances is a well known phenomenon (McIlwain, 1959), whilst storage of fat soluble substances in adipose tissue is another factor. With regard to the latter, it is necessary to take this into account when calculating the dose of a particular drug in terms of dosage/weight ratio in that with those drugs not readly absorbed by adipose tissue, calculation of the "lean body weight" may be more accurate for the purpose of comparison between individuals.

Inactivation

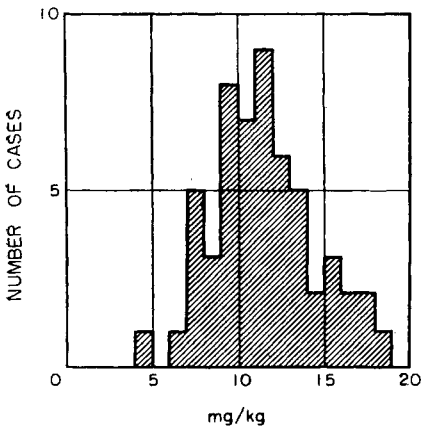
The reduction of drug concentration to a level at which it is no longer pharmacologically active may come about in several ways. As mentioned above this may be just a redistribution and dilution in general body fluids that can occur at the cessation of the inhalation or intravenous injection of a drug (Wilson and Schild, 1959). Secondly, the drug may be eliminated unchanged from the body via kidneys or the lungs. Thirdly, the drug may be actively destroyed through chemical change within the body. As can be readily seen all three may play a part where any individual drug is concerned, and will occur contemporaneously. Thus, individual variations in the rate at which a desired level of drug may be reached may be introduced by variations in the efficiencies of these three mechanisms.

Further to the considerations above, it must be borne in mind that the relationship between drug concentration and drug effect does not remain static in that, at least for some substances, the body starts to adapt to their presence even as their concentration within the body may be building up. For example, it has been found that the deterioration of performance engendered by ethyl alcohol is, for the same concentration of alcohol within the blood, less when the blood alcohol is falling than when it is rising (Drew, Colquhoun and Long, 1959).

It is also interesting to note that whilst the body may appear to demonstrate a form of adaption occurring during a single dosage of a particular drug, some drugs produce a considerable tolerance to the drug when given in repeated doses. Moreover, a cross-tolerance between one drug and another can often occur (Wilson and Schild, 1959).

In the above paragraphs, some of the factors involved in attaining a particular level of drug in response to dosage have been briefly considered. Attention will now be turned to the relationship between magnitude of dosage (and

indirectly to level of drug acting upon the "target organ") and drug "response." If we consider a sample populaton of human subjects in terms of the amount of Sodium Amytal needed to produce drowsiness when given intravenously, the number of cases can be plotted against dosage to give a graph of distribution (Fig. 4) which is somewhat skewed. If, on the other hand, this is



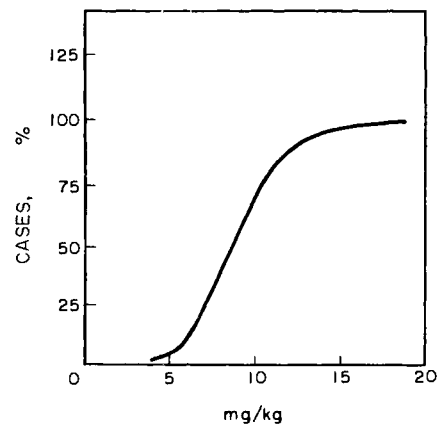


FIG. 4. This shows the distribution of a sample of subjects in terms of the dosage of intravenous Amytal required to produce drowsiness. (After Wilson and Schild. 1959, with permission).

FIG. 5. This shows a cumulative frequency distribution curve based on the sample depicted in Fig. 4. (After Wilson and Schild, 1959, with permission).

plotted in terms of the percentage of the population which would have reached drowsiness at a particular level of drug, a cumulative curve as shown is obtained see Fig. 5). It is interesting to compare this curve with the dose effect curve of a diuretic substance (Fig. 6), but in this case we are dealing not with a population of human subjects but, broadly speaking, with populations of kidney cells whose ability to reabsorb water from the glomerular filtrate has been interfered with. In both these examples, it could be supposed that there is a comparatively small order of interaction between the individual members of the populations. When dose–effect curves are employed it is not unusual to find they occur in this form, whether they are dealing with weight loss engendered by diuretic, or the diminution of size of wheal under the influence of antihistaminic (Fig. 7) (Gaddum 1954). It is usual to plot such curves in terms of log-dose to attain some linearity.

On the other hand, we may ask what is likely to be the effect of a drug on a population where there is a high order of interaction between the members of a population, so that there is a tendency for the drug effect on one member of the population to affect the other members of the population? An example of such an interaction has been demonstrated by the use of amphetamine on a community of mice (Chance, 1947).

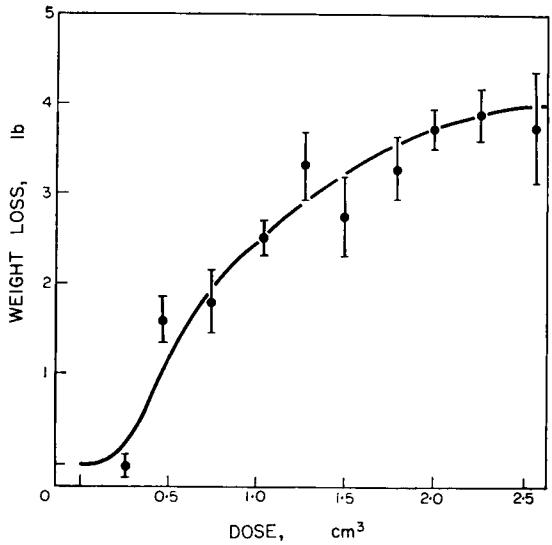


FIG. 6. This shows a curve representing the dose-effect relationship for a diuretic substance. (After Gaddum, 1954, with permission.)

A single mouse, under solitary conditions, exhibits a moderate restlessness under the influence of amphetamine. However, if a community of mice are given amphetamine, those mice most affected by the drug exhibit a contagious restlessness which excites other members of the community so that resultant excitement is much greater than with segregated individuals.

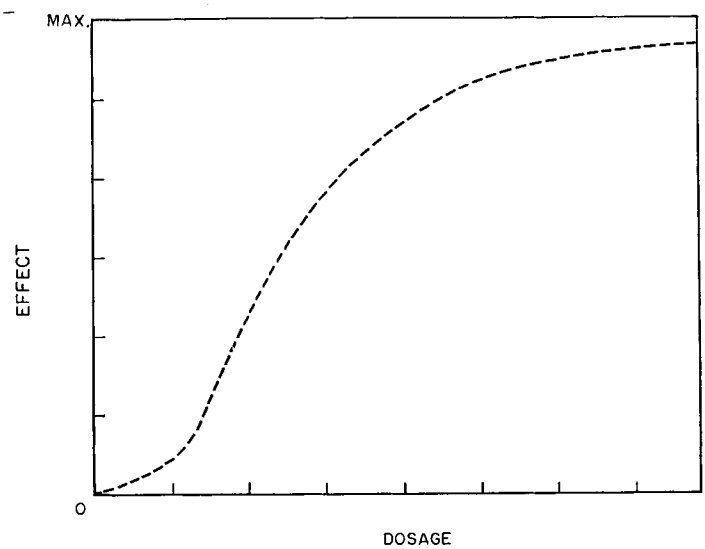


FIG. 7. Diagrammatic representation of the usual type of dose/effect curve found with a variety of pharmacologic agents.

In dealing with the central nervous system, one is dealing with a population of neurones which interact extensively with one another, so that one would expect a more progressive trend in terms of drug effect than, say, with a population of kidney cells. Comparison between Fig. 8 and Fig. 9 dealing with decrement of performance at different levels of nitrous oxide would seem to bear this out.

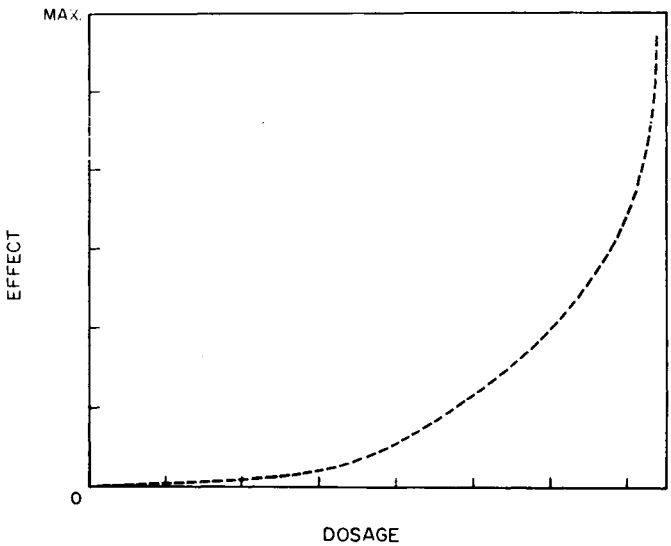


FIG. 8. Diagrammatic representation of the dose/effect relationship between nitrous oxide and decrement in performance on a motor task (cf. Chapter 22).

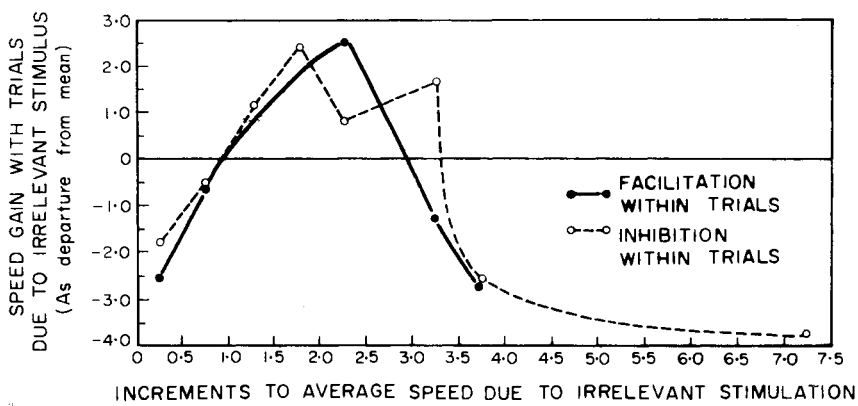


FIG. 8.

Thus in studying the effect of a particular drug, experimentation should be formulated so that the above mentioned factors (viz. (1) the manner in which a pharmacologically active level of drug is attained at the site of action, (2) the ability of the body to adapt to some extent to the drug, over a period of time, and (3) the non-linear relationship between drug level and effect) are borne in mind. The use of appropriate statistical techniques applied to well planned experimentation should enable the difficulties engendered by variability introduced through such factors as these to be overcome.

Conclusion

This chapter has briefly surveyed some of the current body of opinion as to the effect of central nervous system general depressant and stimulant drugs on the central nervous system and the resultant effects on some of the mechanisms underlying response to stimuli. It has considered the reticular formation of the brain as a mechanism for modulating the functioning of other portions of the central nervous system, under the influence of a multitude of factors including both "bombardment" by external stimulation and the pressure of internal biological drives, and has outlined the effects of various drugs on this modulating mechanism.

The general depressant drugs have been depicted as altering the balance between the facilitatory and suppressor functions of the reticular formation so that the functioning of the cortex and peripheral portions of the central nervous system are modified accordingly.

In dealing with the stimulant drugs, the amphetamines have been shown to exert an influence on the tonic portion of the reticular formation so that the effect is one of a general tonic facilitatory influence in opposition to the suppressor mechanisms. This influence is reflected in the appropriate modifications of stimulus–response reactions in various spheres of activity. The drug methylphenidate, in contrast, has been shown as exerting its stimulation not in terms of effect on the tonic activating system but rather on the phasic portion of the reticular formation. Caffeine acts on the phasic portion of the reticular formation and possibly directly on the cerebral cortex itself.

This approach to the effect of drugs on behaviour, whilst utilizing a physiological model tends to be limited by the framework under consideration. The physiologist tends to be interested primarily in the mechanisms underlying observed "behaviour" rather than in the laws governing behaviour as such. To this end he tends to isolate and dissect phenomena so that he may study individual functions which may be at a later date pieced together in a larger pattern. Viewed in this way, behaviour as studied by the behaviourist would be considered as an extension of the functions of all the individual mechanisms which the physiologist studies separately.

The psychologist, on the other hand, in contrast to the from "within–outwards" approach of the physiologist, studies behaviour much more *in vivo* as it were, seeking to determine lawful relationships in the interaction between organism and environment without becoming too deeply involved

in the functional anatomy of the organism, but rather using a system of conceptualizations as a model to relate to the laws of behaviour that he has observed.

It appears reasonable that the more complete a description of a phenomenon that either discipline could render, the more would be the parallelism which could be observed between these two systems of conceptualization.

One of the difficulties in trying to find such a parallelism between the physiological model and the psychological model is that whilst they deal with overlapping areas of observation, the central considerations of each have little common contact. For example, in regard to physiology, the sociopath may differ little from the norm, but in regard to behaviour in a broad sense, he differs markedly.

Nevertheless, as both the psychological description and the neurological description of behaviour become more complete, certain very general coincidences in these descriptions are starting to appear. Most striking is the analogy that can be drawn between the general facilitatory and suppressor functions of the reticular formation and the concepts of central "excitation" and "inhibition" that are used in the psychological model.

BIBLIOGRAPHY and AUTHOR INDEX

Figures in square brackets refer to the pages where the publication in question is referred to.

AARONSON, B.S. Age, intelligence, aphasia and the spiral after-effect in an epileptic population. *J. Clin. Psychol.* 1958, **14,** 18-21. [235]

ACKNER, B. and PAMPIGLIONE, G. Discussion on physiological measurements of "emotional tension". *Proc. Roy. Soc. Med.* 1958, **51,** 76-81. [136, 137, 146, 169, 170]

ACKNER, B. and PAMPIGLIONE, G. An evaluation of the sedation threshold test. *J. Psychosom. Res.* 1959, **3,** 271-281. [146]

ADAMETZ, J.H. Rate of recovery of functioning in cats with rostral reticular lesions. *J. Neurosurg.* 1959, **16,** 85-97. [356]

ADAMS, R. An account of a peculiar optical phenomenon seen after having looked at a moving body. *London and Edinburgh Philosophy Magazine and Journal of Science*, 1834, **5,** 373-374. [240]

ADEY, W.R., SEGUNDO, J.P. and LIVINGSTON, R.B. Corticofugal influences on intrinsic brain stem conduction in cat and monkey. *J. Neurophysiol.* 1957, **20,** 1-16. [144]

ADLER, H.F., BURKHARDT, W.L., IVY, A.C. and ATKINSON, A.J. Effect of various drugs on psychomotor performance at ground level and at simulated altitudes of 18,000 feet in a low pressure chamber. *J. Aviat. Med.* 1950, **21,** 221-236. [67]

AIBA, S. The effects of dexamphetamine, sodium amobarbital and meprobamate on critical frequency of flicker under two different surround illuminations. *Psychopharmacologia*, 1959, **1,** 89-101. [62, 63]

AIBA, S. The effects of stimulant and depressant drugs on the Bidwell phenomenon. *Brit. J. Psychol.* 1960, **51,** 311-318. [51]

ALPERN, M. Metacontrast: historical introduction. *Amer. J. Optom.* 1952, **29,** 631-646. [69, 70]

ALPERN, M. Metacontrast. *J. Opt. Soc. Amer.* 1953, **43,** 648-657. [27, 69, 70]

ALPERN, M. and FARIS, J. Luminance-duration relationship in the electrical response of the human retina. *J. Opt. Soc. Amer.* 1956, **46**, 845-850. [43, 61]

ALPERN, M., FARIS, J., ESKILDSEN, P. and GARNETT, P. Effect of hyperventilation on the human electroretinogram. *Science*, 1955, **121**, 101-102. [61]

AMMONS, C.H. and WEITZ, J. Central and peripheral factors in the phi phenomenon. *J. Exp. Psychol.* 1951, **42,** 327-332. [268]

ARDEN, G., GRANIT, R., and PONTE, F. Phase of suppression following each retinal b-wave in flicker. *J. Neurophysiol.* 1960, **23,** 305-314. [54]

ARDEN, G.B. and WEALE, R.A. Variations of the latent period of vision. *Proc. Roy. Soc. Lond.* 1954, **142** B, 258-266. [54]

ARDUINI, A. and ARDUINI, M.G. Effect of drugs and metabolic alternations on brainstem arousal mechanism. *J. Pharmacol. Exp. Ther.* 1954, **110,** 76. [144, 367]

ASHER, H. Contrast in eye and brain. *Brit. J. Psychol.* 1950, **40,** 187-194. [144]

AX, A.F. The physiological differentiation between fear and anger in humans. *Psychosom. Med.* 1953, **15,** 433-442. [167]

BARD, B and MACHT, M.B. The behavior of chronically decerebrate cats. Symposium: The neurological basis of behavior London: Churchill, 1958. [358]

BARLOW, H.B.. Temporal and spatial summation in human vision at different background intensities. *J. Physiol. (London),* 1958, **141,** 337-350. [42, 48]

BARLOW, H.B. FITZHUGH, R. and KUFFLER, S.W. Change of organization in the receptive fields of the cat's retina during dark-adaptation. *J. Physiol. (London),* 1957, **137,** 338-354. [49]

BARTLEY, S.H. Relation of intensity and duration of brief retinal stimulation by light to the electrical response of the optic cortex of the rabbit. *Amer. J. Physiol.* 1934, **108,** 397-408. [43]

BARTLEY, S.H. *Vision, a Study of Its Basis.* New York: Van Nostrand, 1941. [69, 81]

BAUMGARDT, E and SEGAL, J. Facilitation et inhibition — paramètres de la fonction visuelle. *L'Ann. Psychol.* 1947, **43/44,** 54-102. [27]

BEEBE-CENTRE, J.G. *The Psychology of Pleasantness and Unpleasantness.* New York: Van Nostrand, 1932. [9]

BEECH, H. R. Psychological problems of solitude and confinement. *Biol. of Space Travel. Inst. of Biol. (London),* 1961, 59-74. [9]

BEECHER, H.K. *Measurement of Subjective Responses.* New York: Oxford Univ. Press, 1959. [9, 324]

BENDER, M.B. The phenomenon of sensory displacement. *Arch. Neurol. Psychiat.* 1951, **65,** 607-621. [75]

BENDER, M.B. *Disorders in Perception.* Springfield: Thomas 1952, [3].

BENDER, M.B. and KAHN, R.L. After imagery in defective fields of vision. *J. Neurol. Neurosurg. Psychiat.* 1949, **12,** 196-204. [204]

BENDER, M.B. and TEUBER, H.L. Phenomena of fluctuation, extinction and completion in visual perception. *Arch. Neurol. Psychiat. Chicago,* 1946, **55,** 627-658. [203, 208]

BENDER, M.B. and TEUBER, H.L. Disorders in perception following cerebral lesions. *J. Psychol.* 1949, **28,** 223-233. [269]

BERG, O. A study of the effect of evipan on flicker fusion intensity in brain injuries. *Acta Psychiat. et Neurol,* 1949, Suppl No. **58,** 116. [67]

BERGER, D., EVERSON, R., RUTLEDGE, K. and KOSKOFF, J.D. The spiral after-effect in a neurological setting. *J. Consult. Psychol.* 1958, **22,** 249-255. [232]

BERGER, F.M., KLEITZKIN, M. LUDWIG, B., MARJOLIN, S., and POWEL, L.S. Unusual muscle relaxant and analgesic properties of N-isopropyl-2-methyl-2-propyl-1, 3-propanediol dicarbamate (carisaprodol). *J. Pharmacol. Exp. Ther.* 1959, **127,** 66-73. [324]

BERLYNE, D.E. *Conflict, Arousal* and *Curiosity.* New York: McGraw Hill, 1960. [3, 4, 9, 11]

BERNHARTT, M., GELLHORN, E., and RASMUSSEN, A.T. Experimental contributions to problems of consciousness. *J. Neurophysiol.* 1953, **16,** 21-35. [357]

BERRY, W. and IMUS, H. Quantitative aspects of the flight of colors. *Amer. J. Psychol.* 1935, **47,** 449-457. [208]

BIDWELL, S. On subjective colour phenomena attending sudden changes of illumination. *Proc. Roy. Soc. (London)*, 1896, **60,** 368-377. [27, 119]

BIDWELL, S. On negative after-images following brief retinal excitation. *Proc. Roy. Soc. (London)*, 1897, **61,** 268-271. [27, 119]

BIERSDORF, W.R. Critical duration in visual brightness discrimination for retinal areas of various sizes. *J. Opt. Soc. Amer.* 1955, **45,** 920-925. [42]

BIERSDORF, W.R. Luminance-duration relationships in the light-adapted electroretinogram. *J. Opt. Soc. Amer.* 1958. **48,** 413-417. [43, 61]

BILLS, A.G. The influence of muscular tension on the efficiency of mental work. *Amer. J. Psychol.* 1927, **38,** 227-251. [142]

BILLS, A.G. Blocking: a new principle of mental fatigue. *Amer. J. Psychol.* 1931.. **43,** 230-245. [147]

BJERNER, B. Alpha depression and lowered pulse rate during delayed actions in a serial reaction test. *Acta Psychol. Scand.* 1950, **19,** Suppl. 65, pp. 93 [147]

BLOCK, A.M. Expériences sur la vision. *C.R. Soc. Biol.* (8), Paris, 1885, **2,** 493-495. [42]

BOCCI, B. Note ed appunti di tecnica fisiologica, 11-23, 1900. [200]

BOCK, R.H. A simple after-image test — its diagnostic significance in clinical ophthalmology *Amer. J. Ophthal.* 1952, **35,** 537-543. [203, 208]

BONVALLET, M., DELL, P. and HIEBEL, G. Tonus sympathique et activité électrique corticale. *EEG Clin. Neurophysiol.* 1954, **6,** 119-144. [143, 363]

BORD, B. and MACHT, M.B. The behavior of chronically decerebrate cats. Symposium: *The Neurological Basis of Behavior* (Ciba Foundation). London: Churchill, 1958. [355]

BORG, G. and PAHLSTRÖM, H. *The Perception of Muscular Work.* Publ. of the Amer. Res. Library, 1960. [11]

BORSCHKE, A. and HESCHELES, L. Über Bewegungsnachbilder. *Zeit f. Psychologie und Physiologie d. Sinnesorgane* 1901, **27,** 387-398. [210]

BOUDREAU, D. Evaluation of the sedation threshold test. *A.M.A. Arch. Neurol. Psychiat.* 1958, **80,** 771-775. [136, 137]

BOUMAN, M.A. Peripheral contrast thresholds of the human eye. *J. Opt. Soc. Amer.* 1950, **40,** 825-832. [48]

BOUMAN, M. A. Peripheral contrast thresholds for various and different wavelengths for adapting field and test stimulus. *J. Opt. Soc.Amer.* 1952. **42,** 820-831. [42]

BOWDITCH, H.P. and HALL, G.S. Optical illusion of motion. *J. Physiol.* 1881, **3,** 296-301. [210]

BOYNTON, R.M. and TRIEDMAN, M.H. A psychophysical and electrophysiological study of light adaptation. *J. Exp. Psychol.* 1953, **46,** 125-134. [68]

BRADLEY, P.B. The central action of certain drugs in relation to the reticular formation of the brain. *Reticular Formation of the Brain.* London: Churchill, 1958. [371, 373]

BRADLEY, P.B. and ELKES, J. The effects of some drugs on the electrical activity of the brain. *Brain,* 1957, **80,** 77-117. [372]

BRADLEY, P.B. and JEAVONS, P.M. Effect of chloropromazine and reserpine on the sedation and convulsive thresholds in schizophrenic patients. *EEG Clin. Neurophysiol.* 1957, **9,** 661-672. [136]

BRADLEY, P.B. and KEY, B.J. The effect of drugs on arousal responses produced by electrical stimulation of the reticular formation of the brain. *EEG Clin. Neurophysiol.* 1958, **10,** 97-110. [144]

BRAZIER, M.A.B. The action of anaesthetics on the nervous system. *Brain Mechanisms and Consciousness.* Oxford: Blackwell Scientific Publications, 1953. [368]

BRAZIER, M.A.B. Studies of evoked responses by flash in man and cat. *Reticular Formation of the Brain.* London: Churchill, 1958. [359, 367]

BRENGELMANN, J.C. Complex perceptual processes. In Eysenck, H.J., Granger, G.W. and Brengelmann, J.C. *Perceptual Processes and Mental Illness.* Maudsley Monographs, No. 2, 1957, Chapman and Hall. [204]

BRENNER, M.W. An experimental investigation of apparent movement. Ph. D. Thesis, Univer. of London, 1953. [271, 273, 274]

BRENNER, M.W. The developmental study of apparent movement. *Quart. J. Exp. Psychol.* 1957, **9,** 169-174. [270]

BRINDLEY, G.S. The Bunsen-Roscoe law for the human eye at very short durations. *J. Physiol. (London),* 1952, **118,** 135-139. [42]

BRINDLEY, G.S. The discrimination of after-images. *J. Psysiol.* 1959, **147,** 194-203. [206]

BROADBENT, D.E. *Perception and Communication.* London: Pergamon Press, 1958. [1, 108, 147]

BROADBENT, D.E. Psychophysical methods and individual differences in the kinesthetic figural after-effect. *Brit. J. Phychol.* 1961, **52,** 97-104. [22]

BRODAL, A. *The Reticular Formation of the Brain Stem.* The Henderson Trust, 1957. [358]

BRUNSWIK, E. Thing constancy as measured by correlation coefficients. *Psychol. Rev.* 1940, **47.** [288]

BUDDE, E. Über metakinetische Scheinbewegungen und über die Wahrnehmung von Bewegungen. *Archive f. Anat. und Physiologie.* Dubois Reymond, 1884, 127-152. [229, 241]

BURNS, B. DELISLE, ROBSON, J. G. and WELT, P.J.L. The effects of nitrous oxide upon sensory thresholds. *Canad. Anaes. Soc. J.* 1960, **7,** 411-422. [177]

CAMERON, N. *The Psychology of Behavior Disorders.* Boston: Houghton Mifflin, 1947. [316]

CARPENTER, L.F. Extraversion, neuroticism and verbal learning. Unpublished paper, Univer. of Maryland, 1960. [20]

CATTELL, R.B. *Personality and Motivation Structure and Measurement.* London: Harrap, 1957. [158]

CATTELL, R.B. and SCHEIER, I.H. *The Meaning and Measurement of Neuroticism and Anxiety.* New York: Ronald, 1961. [7]

CHANCE, M.R.A. A peculiar form of social behavior induced in mice by amphetamine. *Behavior,* 1947, **1,** 64-70. [376]

CHEATHAM, P.G. Visual perceptual latency as a function of stimulus brightness and contour shape. *J. Exp. Psychol.* 1952, **43,** 369-380. [69, 70, 72, 120]

CIBIS, P. and NOTHDURFT, H. Experimentelle Trennung eines zentralen und eines peripheren Anteils von unbunten Nachbildern. Lokalisation der Leitungsunterbrechung, die bei experimenteller Netzhautanäemie zu temporärer Amaurose Führt. *Flüg. Arch. f. Ges. Physiol.* 1948, **250,** 501-520. [200]

CLARIDGE, G.S. The excitation-inhibition balance in neurotics. In Eysenck, H.J. (Ed.), *Experiments in Personality.* London: Routledge and Kegan Paul, 1960. [16, 134, 140, 145, 146, 235, 236]

CLARIDGE, G.S. Arousal and inhibition as determinants of the performance of neurotics. *Brit. J. Psychol.* 1961, 52, 53-63 [135, 139]

CLARIDGE, G.S. and HERRINGTON, R.N. Sedation threshold, personality, and the theory of neurosis. *J. Ment. Sci.* 1960, **106,** 1568, 1583. [136, 169, 170, 171, 177]

CLASSEN, A. *Über das Schlusserfahren des Sehens.* pp. 59, seq. Rostock 1863. [242]

COBB, S. *Foundations of Neuropsychiatry.* Baltimore: Williams and Wilkins (4th edition), 1948. [354]

COHEN, J., DEARNALEY, E.J. and HANSEL, C.E.M. The risks taken in driving under the influence alcohol. *Brit. Med. J.* 1958, **1,** 1438. [20]

COHEN, S.I. and SILVERMAN, A.J. Psychophysiological investigations of vascular response variability. *J. Psychosom. Res.* 1959, **3,** 185-210. [167]

CONGER, J.J. The effect of alcohol on conflict behavior in the albino rat. *Quart. J. Stud. Alcohol,* 1951, **12,** 1-29. [330, 346]

CONGER, J.J. Reinforcement theory and the dynamics of alcoholism. *Quart. J. Stud. Alcohol,* 1956, **17,** 296-305. [346]

COOK, S., MAVOR, H., and CHAMBERS, W.F. Effects of reticular stimulation in altered adrenal states. *EEG Clin. Neurophysiol.* 1960, **12,** 601. [144]

COOPER, J.B. Some characteristics of negative after images. *J. Gen. Psychol.* 1948, **39,** 217-228. [202, 209]

COSTELLO, C.G. The control of visual imagery in mental disorder. *J. Ment. Sci.* 1957, **103,** 840-849. [197, 252]

COSTELLO, C.G. The effects of meprobamate on perception. I. Apparent movement. *J. Ment. Sci.* 1960a, **106,** 322-325. [273]

COSTELLO, C.G. The effects of meprobamate on perception. II. The visual after-image. *J. Ment. Sci.* 1960b, **106,** 326-330. [140, 213, 217, 222]

COSTELLO, C.G. The effects of meprobamate on perception. III. The spiral after-effect. *J. Ment. Sci.* 1960c, **106,** 331-336. [239, 244]

COSTELLO, C.G. Further observations on the spiral after-effect. *Percept. Mot. Skills,* 1960d. [237, 244]

COSTELLO, C.G. Massed practice on the spiral after affect and the homeostatic nature of excitation-inhibition. *Percept. Mot. Skills,* 1960e, 12, 11-14 [227, 237, 250, 251, 253, 254]

COSTELLO, C.G. The effects of meprobamate on time perception. *J. Ment. Sci.* 1961, **107,** 67-73. [19]

COSTELLO, C. and EYSENCK, H.J. Persistence, personality and motivation. *Percept. Mot. Skills.* 1961, **12,** 169-170. [325]

COSTELLO, C.G. and MCGREGOR, P. The relationships between some aspects of visual imagery and the alpha rhythm. *J. Ment. Sci.* 1957, **103,** 786-795. [293]

COURTS, F.A. Relations between experimentally induced muscular tension and memorization. *J. Exp. Psychol.* 1939, **25,** 235-256. [142]

CRAIK, K.J.W. Origin of visual after-images. *Nature,* 1940, **145,** 512 [200]

CRAWFORD, B.H. Visual adaptation in relation to brief conditioning stimuli. *Proc. Roy. Soc. Lond., Ser. B.,* 1947, **134,** 283-302. [49]

CREED, R.S. The after-image of black. *J. Physiol.* 1931, **73,** 247-266. [206, 208]

CREED, R.S. and GRANIT, R.A. On the latency of negative after-images following stimulation of different areas of the retina. *J. Physiol.* 1928, **66,** 281-298. [206, 211]

CREED, R.S. and HARDING, R.D. Latency of after-images and interaction between the two retino-cerebral apparatuses in man. *J. Physiol.* 1930, **69,** 423-441. [201, 202, 209]

CROOK, M. N. A test of the central factor in visual adaptation. *J. Gen. Psychol.* 1930, **3,** 313-318. [205]

CRUTCHFIELD, R.S. and EDWARDS, W. The effect of a fixated figure on autokinetic movement. *J. Exp. Psychol.* 1949, **39,** 561-568. [255]

CSANK, R.J. Scores of a normal population on four psycho-physical measures of potential use for the evaluation of psychotic disorders. M.A. Thesis, Univer. of Montreal, 1955. [29]

CULBERT, S.S. Directional after-effects following systematic distortion of the visual field. *J. Psychol.* 1954, **37,** 81-93. [293]

CURTIS, H.J. Intra-cortical connections of corpus callosum as indicated by evoked potentials. *J. Neurophysiol.* 1940, **3,** 407-413. [293]

DALE, H.H., GREENWOOD, M., MELLANBY, E., MYERS, C.S. and SHERRINGTON, S.S. *Alcohol: Its Action on the Human Organism.* London: H.M.S.O., 1938. [329, 330, 347]

DALY, D.D. and YOSS, R.E. Electroencephalogram in narcolepsy. *EEG Clin. Neurophysiol.* 1957, **9,** 109-120. [356]

DANIS, P. Modifications de l'électrorétinogramme du rat produites par l'injection intra-artérielle proche de potassium, de vératrine et de narcotiques. *J. Physiol. (Paris)*, 1956, **48**, 479-483. [54]

DANIS, P. Contribution a l'etude électrophysiologique de la rétine. Imprimerie Medicale et Scientifique, Brussels, 1959. [54]

DAVIDS, A., GOLDENBERG, L. and LANFER, M.W. The relation of the Archimides spiral after-effect and the trail making test to brain damage in children. *J. Consult. Psychol.* 1957, **21**, 429-433. [231]

DAY, R.H. On interocular transfer and the central origin of visual after-effects. *Amer. J. Psychol.* 1958, **71**, 784-790. [102, 229]

DAY, R.H., POLLACK, R.H., and SEAGRIM, G.N. Figural after-effects: a critical review. *Aust. J. Psychol.* 1959, **11**, 15-45. [255, 250]

DEATHERAGE, B.H. Figural after-effects and stroboscopic movement. *Amer. J. Psychol.* 1954, **67**, 259-267. [270, 271, 272]

DEATHERAGE, B.H. and BITTERMAN, M.E. The effect of satiation on stroboscopic movement. *Amer. J. Psychol.* 1952, **65**, 108-109. [268, 272, 293]

DELABERRE, E.B. On the seat of optical after-images. *Amer. J. Psychol.* 1889, **2**, 326-328. [200]

DELL, P.C. Humoral effects on the brain stem reticular formation. In Jasper H.H. *et al.* (Eds.), *Reticular Formation of the Brain*. London: Churchill, 1958. [143, 162, 362, 367]

DELL, P.C. Some basic mechanisms of the translation of bodily needs into behavior. In *The Neurological Basis of Behaviour*. Ciba Symposium, 1958, [143, 357]

DEMBER, W.N. and EARL, R.W. Analysis of exploratory, manipulatory and curiosity behaviors. *Psychol. Rev.* 1957, **64**, 91-96. [9]

DESILVA, H.R. An experimental investigation of the determinants of apparent visual movement. *J. Exp. Psychol.* 1926, **37**, 469-501. [294]

DEUTSCH, J.A. A new phenomenon of apparent movement and its after-effect. *Quart. J. Exp. Psychol.* 1950, **2**, 119-123. [242]

DEUTSCH, J.A. A preliminary report on a new auditory after-effect. *Quart. J. Exp. Psychol.* 1951, **3**, 43-46. [255]

DEUTSCH, J.A. The statistical theory of figural after-effects and acuity. *Brit. J. Psychol.* 1956, **47**, 208-215. [242]

DICKEL, H.A., WOOD, J.A., and DIXON, H.H. Electromyographic studies on meprobamate and the working anxious patient. *Ann. N.Y. Acad. Sci.* 1957, **67**, 780-787.

DIETHER, V.G. Taste sensitivity to compounds of homologous series. *Amer. J. Physiol.* 1951, **165**, 247-250. [330]

DIMASCIO, A. and RINKEL, M. Prediction of clinical effectiveness of psychopharmacologic agents from "Drug Action Profiles" in normal human subjects. In Wortis (Ed.), *Biological Psychology*, Vol. 2. Grune and Stratton, 1960. [213]

DODGE, R. Theories of inhibition. *Psychol. Rev.* 1928, **33**, 106-127, 167-187. [1]

DODGE, R. *Human Variability*. New Haven: Yale Univ. Press, 1931. [1]

DODGE, R.C. and BENEDICT, F.G. *The psychological effects of alcohol*. Washington: Carnegie Institute of Washington, 1915. [330]

DOUGLAS, A.G. A tachistoscopic study of the order of emergence in the process of perception. *Psychol. Monogr.* 1947, **61,** No. 287. [69]

DOWNEY, J.E. An experiment on getting an after-image from a mental image. *Psychol. Rev.* 1901, **8,** 42-55.

DREW, G.C., COLQUHOUN, W.P. and LONG, H.A. *Effect of small doses of alcohol on a skill resembling driving.* MRC Memorandum No. 38, London: H.M.S.O., 1959. [20, 131, 375]

DUFFY, E. The measurement of muscular tension as a technique for the study of emotional tendencies. *Amer. J. Psychol.* 1932, **44,** 146-162. [142]

DUFFY, E. Emotion: An example of the need for reorientation in psychology. *Psychol. Rev.* 1934, **41,** 239-243. [142]

DUFFY, E. An explanation of "emotional" phenomena without the use of the concept "emotion." *J. Gen. Psychol.* 1941, **25,** 283-293. [142]

DUFFY, E. The psychological significance of the concept of "arousal" or activation. *Psychol. Rev.* 1957, **64,** 265-275. [142]

DUNCAN, C.P. On the similarity between reactive inhibition and neural satiation. *Amer. J. Psychol.* 1956, **69,** 227-235. [104, 257]

DUNCAN, C.P. Visual figural after-effects as a function stimulation interval and decay time. *Percept. Mot. Skills,* 1958, **8,** 203-206. [256]

DUNCAN, D.B. Multiple range and multiple F tests. *Biometrics,* 1955, **11,** 1 42. [66]

DUNLAP. K. Light spot adaptation. *Amer. J. Physiol.,* 1921, **55,** 201-211. [205]

DVORAK Versuche über die Nachbilder von Reizveränderungen. *Sitzberichte d. Wiener Akademie d. Wissenschaften,* 1870, *B.* **61,** Abt. 11, 257-262. [228, 240]

DWORKIN, S., BOURNE, W. and RAGINSKY, B.B. Changes in conditioned responses brought about by anaesthetics and sedatives. *Canad. Med. Ass. J.* 1937, **37,** 136-139. [330]

DWORKIN, S., RAGINSKY, B.B. and BOURNE, W. Action of anaesthetics and sedatives upon the inhibited nervous system. *Anaesth. Analges.* 1937, **16,** 235-240. [330]

EASON, R.G. and WHITE, C.T. Relationship between muscular tension and performance during rotary pursuit. *Percept. Mot. Skills,* 1960, **10,** 199-210. [142]

EASTERBROOK, J.A. The effect of emotion on cue utilization and the organization of behavior. *Psychol. Rev.* 1959, **66,** 183-201. [329]

EASTERBROOK, J.A. A study of the effect of prior photic stimulation in facilitation and inhibition of escape from successive electric stimuli by rats treated with water or ethand. Ph.D. Thesis, Univer. of London, to be presented [329, 330, 333, 338]

EBBECKE, U. *Ar. Ges. Physiol.* 1929, **221,** 160-212. [209]

ECCLES, J. C. *The Neurophysiological Bases of Mind.* Oxford: Clarendon Press, 1953. [355]

ECCLES, J.C. *The Behavior of Nerve-cells.* CIBA Foundation Symposium on the Neurological Basis of Behaviour. London: Churchill, 1958. [293]

EDWARDS, A.L. *Statistical Analyses.* New York: Rinehart, 1946. [181]

EGGLETON, M. Grace. The diuretic action of alcohol on man. *J. Physiol.* 1942, **101,** 172-191. [330]

EHRENSTEIN, W. Versuche über die Beziehungen zwischen Bewegungs- und Gestaltwahrnehmungen. *Zsch. f. Psychol.* 1925, **96,** 305-352. [229, 239]

ELKES, J. Drug effects with relationship to receptor specificity within the brain. CIBA Foundation Symposium on the Neurological Basis of Behaviour. London: Churchill, 1958. [356]

ELMADJIAN, F., LAMSON, E.T. and NERI, R. Excretion of adrenaline and noradrenaline in human subjects. *J. Clin. Endocrin.* 1956, **16,** 222-234. [144]

EPSTEIN, S. Overinclusive thinking in a schizophrenic and a control group. *J. Consult. Psychol.* 1953, **17,** 384-388. [316]

EULER, C. von and SODERBERG, U. Medullary chemosensitive receptors. *J. Physiol.* 1942, **118,** 545-554. [330]

EXNER, S. Weitere Untersuchungen über die Regeneration in der Netzhaut und über Druckblindheit. *Pflüg. Arch. f. Ges. Physiol.* 1879, **20,** 614-626. [200]

EXNER, S. Einige Beobachtungen über Bewegungsnachbilder. *Zentralblatt. f. Physiologie,* 1888, **1,** 135-140. [200, 229, 230, 242]

EXNER, S. *Z. Psy. Phys. Sinnes,* 1896, **xii,** 313. [220]

EYSENCK, H.J. "Type" factors in aesthetic judgements. *Brit. J. Psychol.* 1941, **31,** 262-270. [12]

EYSENCK, H.J. *Dimensions of Personality.* London: Routledge and Kegan Paul, 1947. New York: Praeger. [6, 12, 134]

EYSENCK, H.J. *The Scientific Study of Personality.* London: Routledge and Kegan Paul, 1952. New York: Praeger. [6, 303]

EYSENCK, H.J. *The Structure of Human Personality.* London: Methuen, 1953, 1960. [109, 244]

EYSENCK, H.J. *Psychology of Politics.* London: Routledge and Kegan Paul, 1954. [14]

EYSENCK, H.J. A dynamic theory of anxiety and hysteria. *J. Ment. Sci.,* 1955, **101,** 28-51. [132, 135, 169]

EYSENCK, H.J. Critical inhibition, figural after-effects and theory of personality. *J. Abnorm. Soc. Psychol.* 1955, **51,** 94-106. [197, 255]

EYSENCK, H.J. The questionnaire measurement of neuroticism and extraversion. *Riv. di. Psichol.* 1956a, **50,,** 113-140. [162]

EYSENCK, H.J. "Warm up" in pursuit rotor learning as a function of the extinction of conditioned inhibition. *Acta Psychol.,* 1956b, **12,** 349-370. [147]

EYSENCK, H.J. *Dynamics of Anxiety and Hysteria.* London: Routledge and Kegan Paul, 1957a. New York: Praeger. [1, 3, 7, 10, 14, 17, 20, 87, 109, 132, 169, 184, 236, 284, 306, 327]

EYSENCK, H.J. Drugs and personality. I. Theory and methodology *J. Ment. Sci.* 1957b, **103,,** 119 131. [xii, 4, 7, 8, 13, 19, 70, 75, 87, 101, 132, 134, 198, 258, 303, 321, 325, 346]

EYSENCK, H.J. A short questionnaire for the measurement of two dimensions of personality. *J. Appl. Psychol.* 1958, **42,** 14-17. [11]

EYSENCK, H.J. Serial position effects in nonsense syllable learning as a function of interlist rest pauses. *Brit. J. Psychol.* 1959, **50,** 360-362. [11, 12, 14, 20, 21]

EYSENCK, H.J. *Manual of the Maudsley Personality Inventory.* London: Univ. of London Press, 1959. [141, 174, 215, 224, 316, 325, 327]

EYSENCK, H. J. *The Structure of Human Personality.* London: Methuen, 1960a. [5, 6, 7, 12, 16, 166]

EYSENCK, H. J. (Ed.) *Experiments in personality* (2 vols.) London: Routledge and Kegan Paul, 1960b. [New York: Praeger.] [6, 7, 13, 20, 22, 134, 207, 258]

EYSENCK, H.J. Opis i pomiar vsobowvsci. *Psychol. Wychowawcza,* 1960c, **3,** 257-274; 381-401. [8, 142]

EYSENCK, H.J. Levels of personality, constitutional factors and social influences: an experimental approach. *Int J. Soc. Psychiat.* 1960d, VI, 12-24. [3]

EYSENCK, H.J. *Behaviour Therapy and the Neuroses.* London: Pergamon, 1960e. [14]

EYSENCK, H.J. *Handbook of Abnormal Psychology.* London: Pitman, 1960f. New York: Basic Books, 1961. [16]

EYSENCK, H.J. Drug postulates, theoretical deductions and methodological considerations. In Miller, J.G. and Uhr, L. (Eds.) *Drugs and Behavior.* New York: Wiley, 1960g. [64, 87, 92, 115, 314]

EYSENCK, H.J. Symposium: The development of moral values in children. III. The contribution of learning theory. *Brit. J. Educ. Psychol.* 1960h, **30,** 11-21.

EYSENCK, H.J. Objective psychological tests and the assessment of drug effects. *Int. Rev. Neurobiol.,* 1960i, **2,** 333-384. [24, 158, 236]

EYSENCK, H.J. The concept of statistical significance and the controversy about "one-tailed" tests. *Psychol. Rev.* 1960j, **67,** 269-271. [327]

EYSENCK, H.J. Psychosis, drive and inhibition: a theoretical and experimental account. *Amer. J. Psychiat.* 1961, **118,** 198-204. [4]

EYSENCK, H.J. Reminiscence, drive and personality—revision and extension of a theory. *Brit. J. Soc. Clin Psychol.* 1962, **1,** 127-140. [13, 22, 327]

EYSENCK, H.J. and AIBA, S. Drugs and personality. V. The effects of stimulant and depressant drugs on the suppression of the primary visual stimulus. *J. Ment. Sci.* 1957, **103,** 661-665. [19, 38, 49, 52, 56, 60, 61, 119]

EYSENCK, H.J., CASEY, S. and TROUTON, D.S. Drugs and personality. II. The effect of stimulant and depressant drugs on continuous work. *J. Ment. Sci.* 1957, **103,** 645-649. [19, 140]

EYSENCK, H.J. and CLARIDGE, G.S. The position of hysterics and dysthymics in a two-dimensional framework of personality description. *J. Abnorm. Soc. Psychol.* 1962, **64,** 46-55. [12, 162]

EYSENCK, H.J. and EASTERBROOK, J.A. Drugs and personality. VI. The effects of stimulant and depressant drugs upon body sway (static ataxia). *J. Ment. Sci.* 1960a, **106,** 831-834. [19, 234, 264]

EYSENCK, H.J. and EASTERBROOK, J.A. Drugs and personality. VII. The effects of stimulant and depressant drugs upon pupillary reactions. *J. Ment. Sci.* 1960b, **106,** 835-841. [19, 132, 236]

EYSENCK, H.J. and EASTERBROOK, J.A. Drugs and personality. VIII. The effects of stimulant and depressant drugs on visual after-effects of a rotating spiral. *J. Ment. Sci.* 1960c, **106,** 842-844. [19]

EYSENCK, H.J. and EASTERBROOK, J.A. Drugs and personality. IX. The effects of stimulant and depressant drugs upon visual figural after-effects. *J. Ment. Sci.* 1960d, **106,** 845-851. [22]

EYSENCK, H.J. and EASTERBROOK, J.A. Drugs and personality. X. The effects of stimulant and depressant drugs upon kinaesthetic figural after-effects. *J. Ment. Sci.* 1960e, **106,** 852-854. [19]

EYSENCK, H.J. and EASTERBROOK, J.A. Drugs and personality. XI. The effects of stimulant and depressant drugs upon auditory flutter fusion. *J. Ment. Sci.* 1960f, **106,** 855-857. [22]

EYSENCK, H.J. and EYSENCK, S.B.G. Reminiscence on the spiral after-effect as a function of length of rest pause and number of pre-rest trials. *Percept. Mot. Skills*, 1960, **10,** 93-94. [212]

EYSENCK, H.J. and EYSENCK, S.B.G. The classification of drugs according to their behavioural effects: a new method. In: Eysenck, H.J. (Ed.) *Experiments in Personality.* London: Routledge and Kegan Paul, 1960. [24, 238]

EYSENCK, H.J., GRANGER, G.W. and BRENGELMANN, J.C. *Perceptual Processes and Mental Illness.* Maudsley Monographs No. 2. London: Chapman and Hall, 1957. [19]

EYSENCK, H.J., HOLLAND, H. and TROUTON, D.S Drugs and personality III. The effects of stimulant and depressant drugs on visual after-effects. *J. Ment. Sci.* 1957, **103,** 650-655. [19, 21, 233, 235, 236, 238]

EYSENCK, H.J., HOLLAND, H. and TROUTON, D.S. Drugs and personality. IV. The effects of stimulant and depressant drugs on the rate of fluctuation of a reversible perspective figure. *J. Ment. Sci.* 1957, **103,** 656-660. [19, 21, 233, 235, 236, 238, 305]

EYSENCK, H.J. and MAXWELL, A.E. Reminiscence as a function of drive. *Brit. J. Psychol.* 1961, **52,** 43-52.

EYSENCK, H.J., TARRANT, M., ENGLISH, L., and WOOLF, M. Smoking and personality. *Brit. Med. J.* 1960, **1,** 1456-1460. [11]

EYSENCK, S.B.G. An experimental study of psychogalvanic reflex responses of normal, neurotic and psychotic subjects. *J. Psychosom. Res.* 1956, **1,** 258-272. [325]

EYSENCK, S.B.G. Personality and pain assessment in childbirth, of married and unmarried mothers. *J. Ment. Sci.* 1961, **107,** 417-430. [11]

EYSENCK, S.B.G. and EYSENCK, H.J. Rigidity as a function of introversion and extraversion: a study of married and unmarried mothers. *Int. J. Soc. Psychiat.* 1962, **8,** 180-184.

EYSENCK, S.B.G., EYSENCK, H.J., and CLARIDGE, G.S. Dimensions of personality, psychiatric syndromes, and mathematic models. *J. Ment. Sci.* 1960, **106,** 581-589. [134]

FECHNER, G.T. *Poggendorf's Ann.* 1840, **50,** 193-221; 427-465. [209]
FELDBERG, W. A physiological approach to the problem of general anaesthesia and of loss of consciousness. *Brit. Med. J.* 1959, **11,** 771-782. [356, 357, 358, 367]
FELSINGER, J.N., LASAGNA, L. and BEECHER, H.K. The persistence of mental impairment following a hypnotic dose of a barbiturate. *J. Pharmacol. Ther.* 1953, **109,** 284-291. [19]
FERRET, P., BARBUT, P. and DUCOS, P. Etudes des rapports entre le teneur du sang en alcool et les aptitudes psychometrices. *Toulouse Med.* 1951, **52,** 385. [330]
FINK, M. Lateral gaze nystagmus as an index of the sedation threshold. *EEG Clin. Neurophysiol.* 1958, **10,** 162. [137, 173]
FISCHER, E. The counteraction of weak concentrations of azide on the depressing effect of alcohol on the oxygen consumption of brain slices. In Himwich, H.E. (Ed.) *Alcoholism* Washington: American Association for the Advancement of Science, 1957. [330]
FISICHELLI, V.R. Reversible perspective in Lissajous figures: some theoretical considerations. *Amer. J. Psychol.* 1947, **60,** 240-249. [21]
FOULDS, G.A. The relative stability of personality measures compared with diagnostic measures. *J. Ment. Sci.* 1959, **105,** 783-787. [161]
FOULDS, G.A. and CAINE, T.M. Psychoneurotic symptom clusters, trait clusters, and psychological tests. *J. Ment. Sci.* 1959, **104,** 722-731. [161]
FOX, B.H. Figural after-effects: 'satiation', and 'adaptation'. *J. Exp. Psychol.* 1951, **42,** 317-326. [293]
FRANKS, C.M. Conditioning and personality: a study of normal and neurotic subjects. *J. Abnorm. Soc. Psychol.* 1956, **52,** 143-150. [14, 134]
FRANKS, C.M. Personality factors and the rate of conditioning. *Brit. J. Psychol.* 1957, **48,** 119-126. [14, 132]
FRANK, C.M. and LAVERTY, S.G. Sodium amytal and eyelid conditioning. *J. Ment. Sci.* 1955, **101,** 654-663. [19, 132, 315]
FRANKS, C.M. and TROUTON, D.S. Effects of amobarbital sodium and dexamphetamine sulfate on the conditioning of the eyeblink response. *J. Comp. Physiol. Psychol.* 1958, **51,** 220-222. [19, 132, 315]
FRANKS, V. An experimental study of conditioning and learning in mental defectives. Unpublished Ph. D. Thesis: Univer. of London, 1959. [314]
FRANKS, V. and FRANKS, C.M. Conditionability in defectives and normals as related to intelligence and organic deficit. *Proc. London Conf. Sci. Study ment. Def.* 1962, 577-583. [14]
FRANZ, S.I. After images. *Psychol. Rev. Mon. Suppl.* 1899, **3,** No. 2. [208]
FREEMAN, E. Personal Communication, 1959. [234]
FREEMAN, E. and JOSEY, W.E. Quantitative visual index to memory impairment. *Arch. Neurol. Psychiat.* 1949, **62,** 794-797. [234]
FREEMAN, G.L. The relationship between performance level and bodily activity level. *J. Exp. Psychol.* 1940, **26,** 602-608. [142]
FREEMAN, G.L. *The Energetics of Human Behaviour.* Ithaca, N. Y.: Cornell Univ. Press, 1948. [142, 167, 329]

FRENCH, J.D. Corticifugal connections with the reticular formation. In Jasper, H.H. *et al.* (Eds.) *Reticular Formation of the Brain.* London: Churchill, 1957. [144]

FRENCH, J.D., HERNÁNDEZ PÉON, R., and LIVINGSTON, R.B. Projections from cortex to cephalic brain stem (reticular formation) in monkey. *J. Neurophysiol.* 1955, **18,** 74-95. [144]

FRENCH, J.D., VERZEANO, M., and MAGOUN, H. W. A neural basis for the anaesthetic state. *A.M.A. Arch. Neurol. and Psychiat.* 1953, **69,** 519. [144]

FRY, G.A. and ROBERTSON, V.M. After-images observed in complete darkness. *Amer. J. Psychol.* 1937, **49,** 265-276. [202]

FUNKENSTEIN, D.H., GREENBLATT, M., and SOLOMON, H.C. An autonomic nervous system test of prognostic significance in relation to electroshock treatment. *Psychosom. Med.* 1952, **14,** 347-362. [146]

FUNKENSTEIN, D.H., KING, D.H., and DROLETTE, M. The direction of anger during a laboratory stress-inducing situation. *Psychosom. Med.* 1954, **16,** 404-413. [167]

FUSTER, J.M. Tachistoscopic perception in monkeys. *Fed. Proc.* 1957, **16,** 43. [144]

FUSTER, J.M. Subcortical effects of stimulation of brain stem on tachistoscopic perception. *Science,* 1958, **127,** 150. [364]

GADDUM, J.H. Clinical pharmacology. *Proc. Roy. Soc. Med.* 1954, **47,** 195-204. [376, 377]

GALAMBOS, R. Neural mechanisms of audition. *Physiol. Rev.* 1954, **34,** 497. [360]

GALAMBOS, R. Suppression of auditory nerve activity by stimulation of efferent fibres to cochlea. *J. Neurophysiol.* 1956, **19,** 424. [144, 360]

GALLEGO, A. On the effect of ethyl alcohol upon frog nerve. *J. Cell. Comp. Physiol.* 1948, **31,** 97-106. [330]

GALLESE, A.S. Spiral after-effect as a test of organic brain damage. *J. Clin. Psychol.* 1956, **12,** 254-258. [230, 232, 233, 236]

GARRETT, E.S., PRICE, A.C. and DEABLER, A. Diagnostic testing for cortical brain impairment. *A.M.A. Arch. Neurol. Psychiat.* 1957, **77,** 223-225. [231, 232]

GASTAUT, H. The role of the reticular formation in establishing conditioned reactions. In Jasper, H.H. *et. al.* (Eds.), *Reticular Formation of the Brain.* London: Churchill, 1957. [145]

GATES, L.W. The after-effect of visually observed movement. *Amer. J. Psychol.* 1934, **46,** 34-46. [242]

GELDREICH, E.W. Some physiological concomitants of mental work. *Psychol. Monogr.* 1953, **67,** No. 8, 1-28. [142]

GELLHORN, E. *Physiological Foundations of Neurology and Psychiatry.* Minneapolis: Univ. of Minnesota Press, 1953. [160]

GELLHORN, E. *Autonomic Imbalance and the Hypothalamus.* Minneapolis: Univ. of Minnesota Press, 1957. [167]

GELLHORN, E. and SPIERMAN, I. The influence of hyperpnea and of variations of the O_2 and CO_2 tension in the inspired air upon after-images. *Amer. J. Physiol.* 1935, **112,** 620-626. [203]

GENGERELLI, J.A. Apparent movement in relation to homonymous and heteronymous stimulation of the cerebral hemispheres. *J. Exp. Psychol.* 1948, **38,** 592-599. [268]

GEORGE, F.H. On the theory of the figural after-effect. *Canad. J. Psychol.* 1953, **7,** 167-171. [242]

GEREBETZOFF, M.A. Les connexions thalamostrices. Le noyan parafasciculaire et le centre median. *J. Belge Neurol. Psychiat.* 1940, **40,** 407-416. [212]

GERNANDT, B. Die Einwirkung von Alkohol auf die Atmung bei Katzen mit Intaktem und Denerviertem Sinus. *Acta Physiol. Scand.* 1943, **6,** 233-239. [330]

GHOSH, J.J. and QUASTEL, J.H. Narcotics and brain respiration. *Nature,* 1954, **174,** 28-31. [330]

GIBSON, J.J. Adaptation, after-effect and contrast in the perception of curved lines. *J. Exp. Psychol.* 1933, **16,** 1-31. [255]

GIBSON, J.J. Adaptation with negative after-effect. *Psychol. Rev.* 1937, **44,** 222-244. [255, 256]

GIBSON, J.J. and RADNER, M. Adaptation, after-effect and contrast in the perception of tilted lines: 1. Quantitative studies. *J. Exp. Psychol.* 1937, **20,** 453-467. [255, 256]

GILBERSTADT, H., SCHEIN, J.D., and ROSEN, A. Further evaluation of the Archimedes spiral after-effect. *J. Consult. Psychol.* 1958, **22,** 243-248. [232]

GLANZER, M. Curiosity, exploratory drive, and stimulus satiation. *Psychol. Bull.* 1958, **55,** 302-315. [9]

GOLDBERG, L.R. and SMITH, P.A. The clinical usefulness of the Archimedes spiral in the diagnosis of organic brain damage. *J. Consult. Psychol.* 1958, **22,** 155-157. [232]

GOLDSTEIN, A.G. On the after-effects of the waterfall and spiral illusions. *Amer. J. Psychol.* 1958, **71,** 608-609.

GOLLIN, E.S. and BRADFORD, N. Faulty communication and the spiral after-effect: a methodological critique. *J. Abnorm. Soc. Psychol.* 1958, **57,** 122-123. [234]

GOOCH, R.N. and SLATER, P. A mathematical analysis of motor decrement due to increasing concentrations of nitrous oxide. To appear. [181]

GRAHAM, C.H. Visual perception. In Stevens, S.S. (Ed.), *Handbook of Experimental Psychology.* New York: Wiley, 1951. [291, 294]

GRAHAM, C.H. and KEMP. E.H. Brightness discrimination as a function of the duration of the increment in intensity. *J. Gen. Physiol.* 1938, **21,** 635-650. [42, 48]

GRAHAM, C.H. and MARGARIA, R. Area and intensity relation in the peripheral retina. *Amer. J. Physiol.* 1935, **113,** 299-305. [42]

GRANIT, R. On inhibition in the after-effect of seen movement. *Brit. J. Psychol.* 1928, **19,** 147-157. [243]

GRANIT, R. *Sensory Mechanism of the Retina.* London: Oxford Univ. Press, 1947. [61, 62, 68, 102, 103, 105]

GRANIT, R. Centrifugal and antidromic effects on ganglion cells of retina. *J. Neurophysiol.* 1955, **18,** 388-411. [360]

GRANIT, R. *Receptors and Sensory Perception.* New Haven, Conn.: Yale Univ. Press, 1955. [68]

GRANIT, R., HOWENTHAL, T., and UOTI, A. On the latency of negative after-images in relation to brightness of stimulus. *Saertryk Act. Opth.* 1930, **8,** 137-154. [68]

GRENNELL, R.G. Some effects of alcohols on the central nervous system. In Himwich, H.E. (Ed.) *Alcoholism.* Washington: Amer. Ass. Adv. Sci., 1957. [330]

GRIEW, S. and LYNN, R. Construct "reactive inhibition" in the interpretation of age changes in performance. *Nature,* 1960, **186,** No. 4719, 182. [8]

GRIFFITH, B.C. and SPITZ, H.H. Some observations on the spiral after-effect. *Amer. J. Psychol.* 1959, **72,** 139-140. [212]

GRINDLEY, G.C. The relation between the rod and cone mechanisms in the after-effect of seen movement. *J. Physiol.* 1930, **69,** 53-59. [243]

GRINDLEY, G.C. and WILKINSON, R.T. The after-effect of seen movement on a plain field. *Quart. J. Exp. Psychol.* 1953, **5,** 183-184. [229]

GROSS, O. *Die cerebrale Sekundärfunktion.* Leipzig, 1902. [6]

GROSS, O. *Über psychopathologische Mindervertigkeiten.* Leipzig, 1909. [6]

GUILFORD, J.P. Fluctuations of attention with weak visual stimuli. *Amer. J. Psychol.* 1927, **38,** 534-583. [205]

GUILFORD, J.P. *Fundamental Statistics in Psychology and Education.* London: McGraw-Hill, 1956. [173]

HAIZLIP, T.A. and EWING, J.A. Meprobamate habituation. *New. Eng. J. Med.* 1958, **258,** 1181. [92]

HALSTEAD, W.C. *Brain and Intelligence: a Quantitative Study of the Frontal Lobes.* Chicago, Ill.: Univ. of Chicago Press, 1947. [213]

HAMMER, R.E. Temporal factors in figural after-effects. *Amer. J. Psychol.* 1949, **62,** 337-354. [256]

HAHSAN, N.R. *Patterns of Discovery.* Cambridge: Univ. Press, 1958. [24]

HANSEL, C.E.M. Apparent movement and eye movements. *Brit. J. Psychol.* 1953, **44,** 145-155. [286, 294]

HANSEN, A.K. After-image transfer tests in anomalous retinal correspondence. *A.M.A. Arch. Ophthal.* 1954, **52,** 369-374. [202]

HARDING, G.F., GLASSMAN, S.M. and HELZ, W.C. Maturation and the spiral after-effect. *J. Abnorm. Soc. Psychol.* 1957, **54,** 276-277. [234]

HARTLINE, H.K. Intensity and duration in the excitation of single photoreceptor units. *J. Cell. Comp. Physiol.* 1934, **5,** 229-247. [42]

HEBB, D.O. *The Organisation of Behaviour.* New York: Wiley, 1949. [293]

HEBB, D.O. Drives and the CNS (Conceptual Nervous System). *Psychol. Rev.* 1955, **62,** 243-254. [9, 135, 142, 143]

HELMHOLTZ, H. *Handbuch der physiologischen Optik.* 1856, 1866. (Trans. J.P.C. Southall, 1924-1925). [209, 240]

HELSON, H. Adaptation-level as a basis for a quantitative theory of frames of reference. *Psychol. Rev.* 1948, **55,** 297-313. [293]

HENDLEY, C.D., LYNES, T.E. and BERGER, F.M. Effect of meprobamate on electrical activity of thalamus and other subcortical areas. In Himwich, H.E. (Ed.), *Tranquilising Drugs*. American Association for the Advancement of Science, Washington D.C., 1957. [211, 212]

HENRY, C.E. and OBRIST, W. The effect of meprobamate on the EEG. *J. Nerv. Ment. Dis.* 1958, **126,** 268-271. [212]

HENRY, C., OBRIST, W., PORTER, P. and ANGLAS, R. Effect of equanil on the EEG. *Electroencephalog. Clin. Neurophysiol.* 1957, **9,** 172. [356]

HERNANDEZ-PEON, M.D., LAVIN, A., ALCOCER-CUARON, C. and MARCELIN, J.P. Electrical activity of the olfactory bulb during wakefulness and sleep. *EEG. Clin. Neurophysiol.* 1960, **12,** 41-58. [360]

HERNANDEZ-PEON, R., GUZMAN-FLORES, C., ALCARAY, M., and FERNANDEZ-GUARDIOLA, A. Habituation in the visual pathway. *Acta Neurol. Latinoamer.* 1958, **4,** 121. [360]

HERNANDEZ-PEON, R., SCHERRER, H., and JOUVET, M. Modification of electrical activity in the cochlear nucleus during "attention" in unanesthetised cats. *Science*, 1956, **123,** 331. [364]

HERRICK, R.M. Foveal luminance discrimination as a function of the duration of the decrement or the increment in luminance. *J. Exp. Psychol.* 1956, **49,** 437-443. [42]

HERRINGTON, R.N. and CLARIDGE, G.S. The relationship, before and after treatment, between the sedation threshold and the spiral after-effect in schizophrenics. To appear. [159]

HESS, W.R. *Brain Mechanisms and Consciousness*. Oxford: Blackwell Scientific Publications, 1954. [361]

HEUSSE, E. Zwei kleinere Mitteilungen aus dem Gebiete d. physiol. Optik. *Graefe's Archiv. f. Opthalmologie*, 1888, **34,** 127-131. [241]

HEYMANS, C. Action of drugs on carotid body and sinus. *Pharmacol. Rev.* 1955, **7,** 119-142. [330]

HIGGINSON, G. Apparent visual movement and the gestalt. *J. Exp. Psychol.* 1926, **9,** 328-352. [294]

HILDEBRAND, H.P. A factorial study of introversion-extraversion. *Brit. J. Psychol.* 1958, **49,** 1-11. [166]

HIMWICH, H.E. *The Effect of Pharmacologic Agents on the Nervous System*. Williams and Wilkins, 1959. [372]

HINTON, J.M. and MARLEY, E. The effects of meprobamate and pentothal sodium on sleep and mobility during sleep. A controlled trial with psychiatric patients *J. Neurol. Neurosurg. Psychiat.* 1959, **22,** 137-140.

HOAGLAND, H. Some endocrine stress responses in man. In Tanner, J. M. (Ed.), *Stress and Psychiatric Disorder*. Oxford: Blackwell, 1960. [144]

HOLLAND, H.C. The Archimedes Spiral. *Nature*, 1957, **179,** 432-433. [229, 250]

HOLLAND,, H.C. Some determinants of seen movement in the Archimedes spiral. *Acta Psychol.* 1958, **14,** 215-222. [239]

HOLLAND, H.C. Ph. D. Thesis, Univer. of London, 1959. [92, 257]

HOLLAND, H.C. The effects of sodium amylobarbitone and dexamphetamine sulphate on the peripheral visual field. *J. Ment. Sci.* 1960, **106,** 1438-1442. [19, 235, 236]

HOLLAND, H.C. Measures of perceptual functions. In Eysenck, H.J. (Ed.), *Experiments in Personality*. London: Routledge and Kegan Paul, 1960. [140]

HOLLAND, H.C. The effects of depressant drugs on some perceptual processes. In Eysenck, H.J. (Ed.), *Experiments in Personality*. London: Routledge and Kegan Paul, 1960. [140]

HOLLAND, H.C. Drugs and personality. XII. A comparison of several drugs by the flickerfusion method. *J. Ment. Sci.* 1960, **106,** 858-861. [140]

HOLLAND, H.C. and BEECH, H.R. The spiral after-effect as a test of brain damage. *J. Ment. Sci.* 1958, **104,** 466-471. [231, 236]

HOLMBERG, G. and MARTENS, S. Electroencephalographic changes in man correlated with alcohol concentration. *Quart. J. Stud. Alcohol*, 1955, **16,** 411-424. [330]

HOLWAY, A.H. and BORING, E.G. Determinants of apparent visual size with distance variant. *Amer. J. Psychol.* 1941, **54,** [286]

HOPPE, J. Studie zur Erklärung gewisser Scheinbewegungen. *Z. f. Psy. und Phys. d. Sinne.* 1894, **7,** 29-37. [241]

HOVLAND, C.I. Apparent movement. *Psychol. Bull.* 1935, **32,** 755-778. [240]

HSIA, Y. Whiteness constancy as a function of difference in illumination. *Arch. Psychol.* New York, 1943, No. 284. [288]

HUGELIN, A. and BONVALLET, M. Tonus cortical et controle de la facilitation motrice d'origine reticulaire. *J. Physiol. Path. Gen.* 1957, **49,** 1171-1234. [144]

HUGELIN, A., DUMONT, S. and PAILLAS, N. Tympanic muscles and control of auditory input during arousal. *Science*, 1960, **131,** 1371. [328]

HULL, C.L. The influence of caffeine and other factors on certain phenomena of rote learning. *J. Gen. Psychol.* 1935, **13,** 249-264. [17, 20]

HULL, C.L. *Principles of Behaviour*. New York: Appleton-Centure Crofts, 1943. [1, 135, 257, 329, 349]

HUMPHREY, G., DAWE, P.G.M. and MANDELL, B. New high-speed electronic tachistoscope. *Nature*, 1955, **176,** 231-234. [72]

HUNTER, W.S. The after-effect of visual motion. *Psychol. Rev.* 1914, **21,** 245-277. [42]

HUNTER, W.S. Retinal factors in visual after-movement. *Psychol. Rev.* 1915, **22,** 479-489. [42]

HUNTER, W.S. and SIGLER, M. The span of visual discrimination as a function of time and intensity of stimulation. *J. Exp. Psychol.* 1940, **26,** 160-179. [42]

IKEDA, HISAKO and OBONAI, T. The quantitative analysis of figural after-effects. 1. The process of growth and decay of figural after-effects. *Jap. J. Psychol.* 1953a and b, **23,** 246-260; **24,** 59-66. [256]

IKEDA, HISAKO and OBONAI, T. The studies in figural after-effects: IV. The contrast confluence illusion of concentric circles and the figural after-effect. *Jap. Psychol. Res.* 1955, **2,** 17-23. [256]

ILLIG, H., PFLANZ M. and VON UEXKULL, T. Experimentelle Untersuchungen über die Kleinste zeitenkeit (moment) der Optischen Wahrnehmung. *Pflügers Archiv.* 1953, **257,** 121. [271]

INGHAM, J.G. The effect upon monaural sensitivity of continuous stimulation of the opposite ear. *Quart. J. Exp. Psychol.* 1957, **9,** 52-60. [107]

INGHAM, J.G. Variations in cross masking with frequency. *J. Exp. Psychol.* 1959, **58,** 199-205. [3, 107, 108]

INGRAM, W.R. Modification of learning by lesions and stimulation in the diencephalon and related structures. In Jasper, H.H. *et al.* (Eds.), *Reticular Formation of the Brain.* London: Churchill, 1957. [145]

JABLONSKI, W. Optische Untersuchungen am Ganzfeld. IV. Versuche über Nachbilder. *Psychol. Forsch.* 1930, **13,** 146-197. [206]

JACOBSEN, E. and SKAARUP, Y. Experimental induction of conflict behaviour in cats: the effect of some anticholinergic compounds. *Acta Pharm. Tox.* 1955, **11,** 125-134. [330]

JACOBSON, J.H. and GESTRING, G.F. Centrifugal influence upon the electroretinogram. *A.M.A. Arch. Ophthal.* 1958, **60,** 295-302 [68]

JAFFE, R. Influence of cerebral trauma on kinesthetic after-effect. *Amer. Psychologist,* 1951, **6,** 265. [257]

JASMIN, G. and BOIS, P. Effect of centrally acting drugs upon muscular exercise in rats. *Canad. J. Biochem.* 1959, **37,** 417. [373]

JASPER, H.H. Diffuse projection systems: the integrative action of the thalamic reticular system. *EEG. Clin. Neurophysiol.* 1949, **1,** 406-419. [212]

JASPER, HH.. Cortical excitatory state and synchronism in the control of bioelectric autonomous rhythms. *Cold Spring Harbor Symposium Quart. Biol.,* 1958a, **4,** 320-338. [4]

JASPER, H.H. Recent advances in our understanding of the ascending activities of the reticular system. *Reticular formation of the brain.* London: Churchill, 1958b. [363, 364]

JASPER, H.H. and CRUIKSHANK, R. Electroencephalogram: II. Visual stimulation and the after-image as affecting the occipital alpha rhythm *J. Gen. Psychol.* 1937, **28,** 29-48. [207]

JASPER, H.H. and RANEY, E.T. The phi test of lateral dominance. *Amer. J. Psychol.* 1937, **49,** [204]

JELLINEK, E.M. and MCFARLAUD, R.S. Analysis of pyschological experiments in effects of alcohol. *Quart. J. Stud. Alcohol,* 1940, **1,** 272-371. [329]

JOHN, E.R. Higher nervous functions (brain functioning and learning). *Ann. Rev. Physiol.* 1961. [364]

JOHN, E.R. and KILLAM, K.F. Electrophysiological correlates of resistance conditioning in the cat. *J. Pharmacol. Exp. Ther.* 1959, **125,** 252-274. [364]

JOHNSON, D.E., BAUER, R.W. and BROWN, D.R. Visual after phenomena in diagnosis. *J. Consult. Psychol.* 1959, **23,** 90. [203, 205, 208, 233]

JOHNSON, E.P. and BARTLETT, N.R. Effect of stimulus duration on electrical responses of the human retina. *J. Opt. Soc. Amer.* 1956, **46,** 167-170. [43]

JONES, E.E. and BRUNER, J.S. Expectancy in apparent visual movement. *Brit. J. Psychol.* 1954, **45,** 157-165. [234]

JONES, F.N. and BUNTING, E.B. Displacement after-effect in auditory localization. *Amer. Psychologist,* 1949, **4,** 389. [255]

JONES, H. GWYNNE. Learning and abnormal behaviour. In Eysenck, H.J. (Ed.), *Handbook of Abnormal Psychology.* London: Pitman, 1960. [184]

JOUVET, M. and COURJOU, J. Etude neurophysiologique de l'action centrale du chloralhydrate de l'ester methylique de l'acide phenyl piperidyl acétique (Ritaline). *Arch. Int. Pharmacodyn.* 1959, **119,** 189-193. [372]

JUDD, D.B. A quantitative investigation of the Purkinje after-image. *Amer. J. Psychol.* 1927, **38,** 507-533. [205, 210]

JUDD, D.B. Basic correlates of the visual stimulus. In Stevens, S.S. (Ed.), *Handbook of Experimental Psychology.* New York: Wiley, 1951. [205]

JUNG, C.G. *Psychologische Typen.* Zurich: Rascher and Cie, 1921. [6]

KAADA, B.R. and JOHANNESSEN, N.B. Generalised electrocortical activation by cortical stimulation in the cat. *EEG. Clin. Neurophysiol.* 1960, **12,** 567-573. [144]

KAPLAN, S.D. Autonomic visual regulation. *Psychiat. Res. Rep.* 1960a, **12,** 104-118. [29, 119]

KAPLAN, S.D. A visual anolog of the Funkenstein test. *Arch. Gen. Psychiat.* 1960b, **3,** 383-388. [29, 119]

KARN, H.W. Area and the intensity-time relation in the fovea. *Gen. Psychol.* 1936, **14,** 360-369. [42]

KAWI, A.A. The sedation threshold: its concept and use for comparative studies on drug-induced phenomena. *A.M.A. Arch. Neurol. Psychiat.* 1958, **80,** 232-236. [136, 137]

KETY, S.S. and SCHMIDT, C.F. The nitrous oxide method for the quantitative determination of cerebral blood flow in man: theory, procedure and normal values. *J. Clin. Invest.* 1948a, **27,** 476-483. [175, 176, 193]

KETY, S.S. and SCHMIDT, C.F. The effects of altered arterial tensions of carbon dioxide and oxygen on cerebral blood flow and cerebral oxygen consumption of normal young men. *J. Clin. Invest.* 1948, **27,** 484-492. [179]

KILLAM, K.F. and KILLAM, E.K. Drug action on pathways involving the reticular formation. *Reticular Formation of the Brain.* London: Churchill, 1958. [367, 371]

KIMBLE, G.A. An experimental test of a two-factor theory of inhibition. *J. Exp. Psychol.* 1949, **39,** 15-23. [147]

KING, E.E. Differential action of anaesthetics and interneuron depressants on EEG arousal and recruiting responses. *J. Pharmacol. Exp. Ther.* 1956, **116,** 404. [144]

KINOSHITA, T. Zur Kenntnis der negativen Bewegungsnachbilder. *Z. f. Sinnesphys.* 1909, **43,** 420-433. [240]

KINOSHITA, T. Über die Dauer der vegativen Bewegungsnachbilder. *Z.f. Sinnesphysiol.* 1909, **43,** 434-442. [240]

KINZIUS, H. Die Beeinflussung des Blutadrenalinspiegels durch Pervitin, Luminal und Alkohol. *Arbeitsphysiologie, Berl.* 1958, **14,** 243-248. [330]

KITAI, S.T. Centrifugal effects on the electroretinogram of the frog. *Amer. Psychologist,* 1960, **15,** 501. [68]

KLEEMAN, C.A., RUBIN, N.E., LAMDIN, E. and EPSTEIN, F.H. Studies in alcohol diuresis: evaluation of ethyl alcohol as inhibitor of neurohypophysis. *J. Clin. Invest.* 1955, **34,** 448-455. [330]

KLEIN, G.S. and KRECH, D. Cortical conductivity in the brain injured. *J. Pers.* 1952, **21,** 118-148. [3, 203, 208, 257]

KLEINERT, A. Physiol. optische Beobachtungen No. 4. Über Scheinbewegungen. *Pflüger's Archiv. für die Gesamte Physiol.,* 1878, **18,** 573-574. [421]

KLERMAN, G.L., DIMASCIO, A., HAVENS, L.L., and SNELL, J.E. Sedation and tranquillization: A comparison of the effects of a number of psychopharmacologic agents upon normal human subjects. (Unpublished study). 1960. [213]

KLIMPFINGER, S. Über den Einfluss von intenitonaler Einstellung und Übung auf die Gestaltkonstanz. *Arch. Ges. Psychol.* 1933, **88.** [289]

KLING, J.W., WILLIAMS, J.P., and SCHLOSBERG, H. Patterns of skin conductance during rotary pursuit. *Percept. Mot. Skills,* 1959, **9,** 303-312. [142]

KOFFKA, K. *Principles of Gestalt Psychology.* New York: Harcourt, Brace, 1935. [291]

KOGAN, A.B. In *The Moscow Colloquium on Electroencephalography of Higher Nervous Activity.* (Eds.) Jasper, H.H. and Smirnov, G. D. Suppl. 13, *EEG. Clin. Neurophysiol.* 1960. [364]

KŐHLER, W. *Dynamics in Psychology.* New York: Liveright, 1940. [69, 293]

KŐHLER, W. and FISHBACK, J. The destruction of the Müller-Lyer illusion in repeated trials: I. An examination of two theories. *J. Exp. Psychol.* 1950, **40,** 267-281. [293]

KŐHLER, W. and WALLACH, H. Figural after-effects: an investigation of visual processes. *Proc. Amer. Phil. Soc.* 1944, **88,** 269-357. [255, 257, 293]

KOLERS, P.A. and ROSNER, B.S. On visual masking (metacontrast): dichoptic observation. *Amer. J. Psychol.* 1960, **73,** 2-21. [70, 71, 72, 81]

KONORSKI, J. *Conditioned Reflexes and Nervous Organisation.* Cambridge: Univ. Press, 1940. [1]

KORNETSKY, C. Effects of meprobamate, phenobarbital and dextroamphetamine on reaction time and learning in man. *J. Pharmacol. Exp. Therap.* 1958, **123,** 216-219. [213]

KRAGH, U. *The Actual-genetic Model of Perception and personality.* Lund: Gleerup, 1955. [199, 204]

KRAUSKOPF, J. Figural after-effects in auditory space. *Amer. J. Psychol.* 1954, **67,** 278-287. [256]

KRAVKOV, S.V. Colour vision and the autonomic nervous system. *J. Opt. Soc. Amer.* 1941, **31,** 335 337. [29]

KRUGER, L., FELDZAMEN, A.N., and MILES, W.R. Comparative olfactory intensities of the oliphatic alcohols in man. *Amer. J. Psychol.* 1955, **68,** 386-395. [330]

LACEY, J.I. Individual differences in somatic response patterns. *J. Comp, physiol. Psychol.* 1950, **43,** 338-350. [156, 160]

LACEY, J.I. The evaluation of autonomic responses: toward a general solution. *Ann. N. Y. Acad. Sci.* 1956-7, **67,** 125-164. [160]

LACEY, J.I, BATEMAN, D.E. and VAN LEHN, R. Autonomic response specificity. An experimental study. *Psychosom. Med.* 1953, **15,** 8-21. [156]

LACEY, J.I. and LACEY, B.C. Verification and extension of the principle of autonomic response stereotype. *Amer. J. Psychol.* 1958, **71,** 50-73. [156]

LANDIS, C. Determinants of the critical flicker fusion threshold. *Physiol. Rev.* 1954, **34,** 259-286. [75, 104]

LANDIS, C. and ZUBIN, J. The effect of thonzylamine hydrochloride and phenobarbital sodium on certain psychological functions. *J. Psychol.* 1951, **31,** 181-200. [67]

LARABEE, M.G. and PASTERNAK, JEAN M. Selective action of anaesthetics on synapses and axones in mammalian sympathetic ganglia. *J. Neurophysiol.* 1952, **15,** 91-114. [330, 367]

LASHLEY, K.S., CHOW, K.L. and SEMMES, J. An examination of the electrical field theory of cerebral integration. *Psychol. Rev.* 1951, **58,** 123-136. [285, 293]

LAVERTY, S.G. Sodium amytal and extraversion. *J. Neurol. Neurosurg. Psychiat.* 1958, **21,** 50-54. [136]

LAVERTY, S.G. and FRANKS, C.M. Sodium amytal and behaviour in neurotic subjects. *J. Neurol. Neurosurg. Psychiat.* 1956, **19,** 137-143. [315]

LE BEAU, J. *Psycho-chirurgie et fonctions mentales.* Paris: Masson, 1956. [321]

LEE, A.J. *A Synopsis of Anaesthesia* (4th edition) Bristol: John Wright, 1959. [174, 175, 367, 374]

LEEPER, R.W. A motivational theory of emotion to replace "emotion as disorganised response". *Psychol. Rev.* 1948, **55,** 5-21. [142]

LEHMANN, H.E. Preliminary report on a device for the objective measurement of the negative after-image phenomenon. *Science,* 1950, **112,** 199-201. [29, 43, 119]

LEHMANN, H.E. and CSANK, J. Differential screening of phrenotropic agents in man — psychophysiologic test data. *J. Clin. Exp. Psychopath.* 1957, **18,** 222-235. [29, 119]

LEIRI, F. Gibt es einen extralentikularen Akkomodations-mechanismus im Auge? *Gr. Arch. Ophthal.* 1928, **121,** 213-224. [242]

LEIRI, F. Über Optokinetische Hervorgerufene Visuelle Täuschungen. *Gr. Arch. Ophthal.* 1928, **119,** 719-732. [242]

LENKA, C. Toward some interpretation of learning theories: the concept of optimal stimulation. *Psychol. Rep.* 1955, **1,** 27-33. [9]

LESTER, D. and GREENBERG, L.A. The inhalation of ethyl alcohol by men. *Quart. J. Stud. Alcohol*, 1951, **12,** 167-178. [330]

LEVINE, J. and GRAHAM, C.H. The latency of negative visual after-effects as a function of the intensity of illuminations on an adjacent retinal region. *Amer. J. Psychol.* 1937, **49,** 661-665. [222]

LEVINSON, H. *The relation of after-image duration to certain aspects of personality.* Ph. D. Thesis, Univer. of Kansas, 1952. [29, 119, 205, 208]

LILJESTRAND, A. Respiratory reactions elicited from medulla oblongata of the cat. *Acta Physiol. Scand.* 1953, **29,** 323-393. [330]

LILJESTRAND, G. The effects of ethyl alcohol and some related substances on baroceptor and chemoreceptors activity. *Acta Physiol. Scand.* 1953, **29,** 74-82. [330]

LINDSLEY, D.B. In: Stevens, S.S. (Ed.), *Handbook of Experimental Psychology.* London: Chapman and Hall, 1951. [144]

LINDSLEY, D.B. Physiological psychology. *Ann. Rev. Psychol.* 1956, **7,** 323-348. [143]

LINDSLEY, D.B. Psychophysiology and motivation. In Jones, M.R. (Ed.), *Nebraska Symposium on Motivation.* Lincoln: Univer. of Nebraska Press, 1957a. [143]

LINDSLEY, D.B. The reticular system and perceptual discrimination. In Jasper, H.H. *et al.* (Eds.) *Reticular Formation of the Brain.* London: Churchill, 1957b. [69, 145]

LINDSLEY, D.B. and EMMONS, W.H. Perceptual blanking, evoked potentials. and perception time. *EEG Clin. Neurophysiol.*, 1958, **10,** 359. [69]

LINDSLEY, D.B. and EMMONS, W.H. Perception time and evoked potentials *Science*, 1958, **127,** 1061. [69]

LINKE, P.F. *Grundfragen der Wahrnehmungslehre.* Munich: Ernst Reinhardt, 1929. [294]

LITCHFIELD, H.R. Clinical evaluation of meprobamate in disturbed and pre-psychotic children. *Ann. N.Y. Acad. Sci.* 1957, **67,** 828-831. [92]

LIVINGSTONE, R.B. Central control of afferent activity. In *Reticular formation of the brain.* London: Churchill, 1958. [363]

LIVSON, N.H. After-effects of prolonged inspection of apparent movement. *Amer. J. Psychol.* 1953, **66,** 365-376.

LONDON, P. and BRYAN, J.H. Theory and research on the clinical use of the Archimedes spiral. *J. Gen. Psychol.* 1960, **62,** 113-125. [237, 238]

LOOMIS, T. A. The effect of alcohol on myocardial and respiratory function. *Quart. J. Stud. Alcohol*, 1952, **13,** 561-570. [332]

LOTZE, H. *Medizinische Psychologie.* Leipzig, 1852. p. 443. [240]

LUDVIGH, E. Perception of contour: 1. Introduction. *U.S. Naval Sch. Aviation Med.* 17h August, 1953, Rep. No. 4. [69]

LYNN, R. Reversible perspective as a function of stimulus-intensity. *Amer. J. Psychol.* 1961, **74,** 131-133. [21]

LYNN, R. Risk-taking, arousal, introversion and neuroticism. To appear, 1962. [11]

LYNN, R. Individual differences in extraversion and the spiral after-effect. (Unpublished study). 1959. [235, 236, 238]

LYNN, R. and BUTLER, J. Extraversium and the arousal jag: *Brit. J. Sc. clin. Psychol.*, 1962. **1,** 150-151 [11]

LYNN, R. and EYSENCK, H.J. Tolerance for pain, extraversion and neuroticism. *Percept. Mot. Skills*, 1961. **12,** 161-162. [11]

McCLELLAND, P.C., ATKINSON, J.W., CLARK, R.A. and LOVELL, E.L. *The Achievement Motive.* New York: Appleton-Century-Crofts, 1953. [9]

McDOUGALL, W. Some new observations in support of Thomas Young's theory of light and colour vision. *Mind N.S.* 1901, **10,** 52-97, 210-245, 347-382 [205, 206, 209]

McDOUGALL, W. The sensations excited by a single momentary stimulation of the eye. *Brit. J. Psychol.* 1904, **1,** 78-113. [200]

McDOUGALL, W. *Outline of Abnormal Psychology.* London: Methuen, 1924. [20]

McDOUGALL, W. The chemical theory of temperament applied to introversion and extraversion. *J. Abnorm. Soc. Psychol.* 1929, **24,** 293-309. [17, 131, 246]

McEWEN, P. Figural after-effects. *Brit. J. Psychol. (Monogr. Suppl.* No. 31), 1958. [255, 272, 293]

McFARLAND, R.A., HURVICH, L.M. and HALPERIN, M.H. Effect of oxygen deprivation on the relation between stimulus intensity and the latency of visual after-images. *Amer. J. Physiol.* 1943, **140,** 354-366. [203, 206]

McGEOCH, J.A. and IRION, A.L. *The Psychology of Human Learning.* New York: Longmann, Green, 1952. [1]

McILWAIN, H. *Biochemistry and the Central Nervous System* (2nd edition). London: Churchill, 1959. [365, 366, 367, 370, 375]

McREYNOLDS, P. A restricted conceptualization of human anxiety and motivation. *Psychol. Rep.* 1956, **2,** 293-312. [9]

MACH, E. *Grundlinie d. Lehre v. d. Bewegungsempfindungen.* 1895, pp. 59-65. [241]

MACKWORTH, H. *Researches in the Measurement of Human Performance.* M.R.C. Special Report No. 268. London: H.M.S.O., 1948. [19]

MALMO, R.B. Anxiety and behavioural arousal. *Psychol. Rev.* 1957, **64,** 276-287. [142, 162]

MALMO, R.B. Measurement of drive: an unsolved problem in psychology. In Jones, M.R. (Ed.), *Nebraska Symposium on Motivation.* Lincoln: Univ. of Nebraska Press, 1958. [142]

MALMO, R.B. Activation: a neuropsychological dimension. *Psychol. Rev.* 1959, **66,** 367-386. [142, 354]

MALMO, R.B. and DAVIS, J.F. Physiological gradients as indicants of "arousal" in mirror tracing. *Canad. J. Psychol.* 1956, **10,** 231-238. [142],

MALMO, R.B. and SHAGASS, C. Studies of blood pressure in psychiatric patients under stress. *Psychosom. Med.* 1952, **14,** 82-93. [142]

MARGOLIS, L.H. Pharmacotherapy in psychiatry: a review. *Ann. N. Y. Acad. Sci.* 1957, **66,** 698-718. [92]

MARKS, M.R. A further investigation of the Köhler effect. *Amer. J. Psychol.* 1949, **62,** 62-74. [263, 293]

MARQUIS, D.G., KELLY, E.L., MILLER, J.G., GERARD, R.W. and RAPOPORT, A. Experimental studies of behavioural effects of meprobamate on normal subjects. *Ann. N.Y. Acad. Sci.* 1957, **67,** 701-711. [92, 213]

MARSHALL, C. and WALKER, A.E. The electroencephahолographic changes after hemisphectomy in man. *EEG. Clin. Neurophysiol.* 1950, **2,** 143-156. [355]

MARTIN, I. The effects of depressant drugs on palmar skin resistance and adaptation. In Eysenck, H.J. (Ed.), *Experiments in Personality*. London: Routledge and Kegan Paul, 1960. [3, 4, 19]

MARTIN, I. The effects of depressant drugs on reaction times and "set." In Eysenck, H.J. (Ed.), *Experiments in Personality*. London: Routledge and Kegan Paul, 1960. [20]

MARTIN, I. Variations in skin resistance and their relationship to G.S.R. conditioning. *J. Ment. Sci.* 1960, **106,** 281-287. [20]

MASSERMAN, J.H. and YUM, K.A. An analysis of the influence of alcohol on experimental neuroses in cats. *Psychosom. Med.* 1946, **8,** 37-52. [330, 346, 347]

MATHER, K. *Statistical Analysis in Biology*. London: Methuen, 1951. [335, 336]

MEILI, R. and TOBLER, E. Stroboscopic movement in children. *Arch. Psychol. (Geneve)*, 1931, **23,** 131-156. [270]

MILES, G.H. The formation of projected visual images by intermittent retinal stimulation. II. Apparatus, procedure and results. *Brit. J. Psychol.* 1915, **8,** 93-126. [68]

MILLER, J.G. *The Pharmacology and Clinical Usefulness of Carisaprodol*. Detroit: Wayne State Univ. Press, 1959. [324]

MILLER, N.E. *Learnable Drives and Rewards*. In Stevens, S.S. (Ed.), *Handbook of Experimental Psychology* New York: Wiley, 1951. pp. 435-472. [346]

MILLETTO, G., COLOMB, H. and CARDIARE, G. Action du meprobamate sur l'E.E.G. humain. *EEG. Clin. Neurophysiol.* 1956, **8,** 715. [212]

MIZIAK, H. and LOZITO, C.C. Latency and duration of binocular and monocular after-images. *J. Exp. Psychol.* 1951, **42,** 247-249. [202, 209]

MORSH, J.E. and ABBOTT, H.D. An investigation of after-images. *J. Comp. Psychol.* 1945, **38,** 47-63. [216]

MORUZZI, G. Synchronising influences of the brain stem and the inhibitory mechanisms underlying the production of sleep by sensory stimulation. *The Moscow Colloquium on Electroencephalography of Higher Nervous Activity*. Edited by H.H. Jasper and G.D. Smirnov. Suppl. No. 13 of *EEG Clin. Neurophysiol.* 1960. [362, 363, 365, 367, 368]

MORUZZI, G. and MAGOUN, H.W. Brain stem reticular formation and activation of the EEG. *EEG Clin. Neurophysiol.* 1949, **1,** 455-473. [143, 367]

MOUNIER, M. and TISSOT, R. Correlated effects in behavior and electrical brain activity evoked by stimulation of the reticular system, thalamus and rhinencephalon in the conscious animal. Symposium: *The Neurological Basis of Behavior.* (Ciba Foundation). London: Churchill, 1958. [357]

MOWRER, O.H. *Learning Theory and Personality Dynamics.* New York: Ronald, 1950. [14]

MULLER, J. *Handbuch d. Physiol. d. Menschen,* 1840, **2,** 365. Coblenz. [241]

NAGAMATA, H. Contribution to the knowledge of after-images. *Acta Soc. ophthal. Jap.* 1951, **55,** 802-806. [209]

NAGASAWA, A. and OBONAI, T. A study of the relation between the visual-after-image and brain waves. *Jap. J. Psychol.* 1957, **27,** 342-351. [204]

NAQUET, R., REGIS, H., FISCHER-WILLIAMS, M., and FERNANDEZ-GUARDIOLA, A. Variations in the responses evoked by light along the specific pathways. *Brain,* 1959, **83,** 52-56. [315]

NAUTA, W.J.H. and KUPPERS, H.G.J.M. Some ascending pathways in the brain stem reticular formation. In. *Reticular Formation of the Brain* London: Churchill, 1958. [358]

NEBYLITSYN, V.D., ROZHDESTVENSKAYA, V.I. and TEPLOV, B.M. Concerning the interrelation between absolute sensitivity and strength of the nervous system. *Quart. J. Exp. Psychol.* 1960, **12,** 17-25. [184]

NEFF, W.S. A critical investigation of the visual appearance of movement. *Amer. J. Psychol.* 1936, **48,** 1-42. [268]

NEUHAUS, W. Experimentelle Untersuchung der Scheinbewegung. *Arch. Ges. Psychol.* 1930, **75,** 315-458. [294]

NICHOLLS, E.G. The relation between certain personality variables and the figural after-effect. Ph. D. Thesis, Univer. of London:, 1955. [208, 209, 257, 271, 273].

NYMGARD, K. Studies on the sedation threshold. A. Reproductibility and effect of drugs. B. Sedation threshold in neurotic and psychotic depression. *Arch. Gen. Psychiat.* 1959, **1,** 530-536. [136]

OHWAKI, Y. and KIHARA, T. A new research on the so-called "Bocci Image". *Tohoku Psychol. Folia,* 1953, **13,** 157-180. [201]

OPPEL, J.J. Neue Beobachtungen und Versuche über eine eigentümliche noch wenig bekannte Reaktionstätigkeit d. Menschlichen Auges. *Pogg. Ann.* 1856, **99,** 540-561. [241]

OSGOOD, C.E. and HEYER, A.W., Jr. A new interpretation of figural after-effects. *Psychol. Rev.* 1952, **59,** 98-118. [242, 263, 295]

OSWALD, I. After-images from retina and brain. *Quart. J. Exp. Psychol.* 1957, **9,** 88-100. [199, 200]

PAGE, H.A., RAKITA, G., KAPLAN, H.K. and SMITH, N.B. Another application of the spiral after-effect in the determination of brain damage. *J. Consult. Psychol.* 1957, **21,** 89-91. [231, 236]

PARINAUD, H. Du siege cerebral des images accidentelles ou consecutives. *Gazette des Hopitaux*, 1882, **55**, 459 [200]

PATON, W.D.M. The principles of drug action. *Proc. Roy. Soc. Med.* 1960, **53**, 815-820. [375]

PAVLOV, I.P. *Conditioned Reflexes.* London: Oxford Univ. Press, 1927. [1, 17, 252, 284]

PAVLOV, I.P. *Lectures on Conditioned Reflexes.* (W.H. Gantt translation). New York: International Publishers, 1928. [330, 347]

PAVLOV, I.P. Experimental pathology of the higher nervous activity. In *Selected Works.* Moscow: Foreign languages publishing house, 1955. [131]

PAYNE, R.W. and HEWLETT, J.H.G. Thought disorder in psychotic patients. In Eysenck, H.J. (Ed.), *Experiments in Personality.* London: Routledge and Kegan Paul, 1960. [160, 316]

PAYNE, R.W. and HIRST, H.L. Overinclusive thinking in a depressant and a control group. *J. Consult. Psychol.* 1957, **21**, 186-188. [316]

PAYNE, R.W., MATUSSEK, P. and GEORGE, E.I. An experimental study of schizophrenic thought disorder. *J. Ment. Sci.* 1959, **105**, 627-652. [316]

PEREZ-REYES, M. Bioelectrical investigations of pharmacologically induced changes in the level of consciousness. Paper read to the Residents Research Conference, North Carolina Neuropsychiatric Association, October, 1960. [137]

PERMAN, E.S. The effect of ethyl alcohol on the secretion from the adrenal cortex in man. *Acta Physiol. Scand.* 1958, **44**, 241-247. [330]

PERSKY, H., GRINKER, R.R., HAMBURG, D.A., SABSHIN, M.A., KORCHIN, S.J., BASOWITZ, H. and CHEVALIER, J.A. Adrenal cortical function in anxious humans. *A.M.A. Arch. Neurol. Psychiat.* 1956, **76**, 549-558. [144]

PERSKY, H., KORCHIN, S.J., BASOWITZ, H., BOARD, F.A., SABSHIN, M., HAMBURG, D.A. and GRINKER, R.R. Effect of two psychological stresses on adrenocortical function. *A.M.A. Arch. Neurol. Psychiat.* 1959, **81**, 219-226. [144]

PETERMAN, B. *The Gestalt Theory and the Problem of Configuration.* London: Routledge and Kegan Paul, 1932. [294]

PETRIE, A. *Personality and the Frontal Lobes.* London: Routledge and Kegan Paul, 1952. [7, 321]

PETRIE, A. Some psychological aspects of pain and the relief of suffering. *Ann. N.Y. Acad. Sci.* 1960, **86**, 13-27. [11]

PETRIE, A., COLLINS, W., and SOLOMON, P. Pain sensitivity, sensory deprivation, and susceptibility to satiation. *Science*, 1958, **128**, 1431-1433. [163]

PETRIE, A., COLLINS, W. and SOLOMON, P. The tolerance for pain and for sensory deprivation. *Amer. J. Psychol.* 1960, **73**, 80-90. [11, 163]

PFFAFFMAN, C. Taste and smell. In Stevens, S.S. (Ed.), *Handbook of Experimental Psychology.* New York: Wiley 1951. [330]

PFEIFFER, C.C., RIOPELLE, A.J., SMITH, R.P., JENNEY, E.H. and WILLIAMS, H.L. Comparative study of the effect of meprobamate on the conditioned response, on strychnine and pentylenetetrazol thresholds on the normal electroencephalogram and on polysynaptic reflexes. *Amer N. Y. Acad. Sci.* 1957, **67,** 734-745. [212]

PHILBRICK, E.B. The validity of the spiral after-effect as a clinical tool for diagnosis of organic brain pathology. *J. Consult. Psychol.* 1959, **23,** 39-43. [232, 236]

PICKERSGILL, M.J. and JEEVES, M.A. After-effect of movement produced by a rotating spiral. *Nature*, 1958, **182,** 1820. [229, 238, 239, 250]

PIERON, H. *The Sensations.* London: Mueller, 1952. [294]

PLATEAU, J. Vierte Notiz über eine neue sonderbare Anwendung des Verweilers der Eindrücke auf der Netzhaut. *Pogg. Ann.* 1850, **80,** 287-292. [240]

POPOV, C. Contribution a l'étude d'une propriéte repetitive du cerveau. *Experientia*, 1956, **12,** 274-275. [204]

POPOV, N.A. Le conditionnement des images consecutives. *C.R. Acad. Sci. Paris*, 1953, **236,** 744-6. [204]

POPOV, N.A. and POPOV, C. Action de l'alcool sur les images consecutives et leur conditionnement. *C.R. Acad. Sci. Paris*, 1953, **237,** 930-932. [204, 208]

POSER, E. Kinaesthetic figural after-effects as a measure of cortical excitation and inhibition. *Amer. Psychologist*, 1958, **13,** 334-335. [19]

POSER, E. Der figurale After-Effekt als Persoenlichkeitsmerkmal: Theorie und Methodik. Paper read at XV Int. Congr. Exp. Psychol., Bonn, 1960. [11]

POULIOT, S. and MISIAK, H. The measurement of negative after-images in first grade boys and girls. *J. Gen. Psychol.* 1959, **95,** 13-17. [216]

PRICE, A.C. and DEABLER, H.L. Diagnosis of organicity by means of the spiral after-effect. *J. Consult. Psychol.* 1955, **19,** 299-302. [229, 230 234]

PRICE, A.C., GARRETT, E.S., HARDY, M.W. and HALL, H.E. Perception of binaural beats in organic and monorganic patients. *A.M.A. Arch. Neurol. and Psychiat.* 1958, **79,** 214-216. [232]

PURKINJE, J. *Beobachtungen und Versuche zur Physiol. der Sinnes.*, 1825, **11,** p. 60. [240]

PURPURA, D.P. Electrophysiological analysis of psychotogenic drug action. *Arch. Neurol. Psychiat.* 1956, **75,** 122-143. [373]

RACHMAN, S. Psychomotor behaviour and personality with particular reference to conflict. London: Unpublished Ph. D. Thesis, 1961a. [11]

RACHMAN, S. Effect of a stimulant drug on extent of motor responses. *Percept. Mot. Skills*, 1961b, **12,** 186. [11, 19]

RAFFY, A. Variations de l'intensite respiratoire des poissons soumis a des eaux alcoolisées. *C.R. Soc. Biol. Paris*, 1949, **143,** 70-71. [330]

RASMUSSEN, G.L. The olivary peduncle and other fibre projections of the superior olivary complex. *J. Comp. Neurol.* 1946, **84,** 141. [360]

RASMUSSEN, G.L. Further observations on the efferent cochlear bundle. *J. Comp. Neurol.* 1953, **99,** 61. [360]

RATCLIFF, F., MILLER, W.H., and HARTLINE, H.K. Neural interaction in the eye and the interaction of receptor activity. *Ann. N. Y. Acad. Sci.* 1958, **74,** 210-222. [81, 105]

RATOOSH, P. and GRAHAM, C.H. Areal effects in foveal brightness discrimination. *J. Exp. Psychol.* 1951, **42,** 367-375. [48]

RAZRAN, G. The observable unconscious and the inferable conscious in current Soviet psychophysiology. *Psychol. Rev.* 1961, **68,** 81-147. [3, 327]

REDLICH, F.C., CALLAHAN, A. and MENDELSON, R.H. Electroencephalographic changes after eye opening and visual stimulation. *Yale J. Biol. Med.* 1946, **18,** 367-376. [356]

REED, G.F. Audiometric response consistency, auditory fatigue and personality. *Percept. Mot. Skills,* 1961, **12,** 126. [108]

REITAN, R.M. The comparative effect of placebo, and meprobamate on psychologic test performance. *Antibiotic Med. Clin. Therapy,* 1957, **4,** 158-165. [213]

REXROAD, C.N. Eye movements and visual after-images. *Amer. J. Psychol.* 1928, **40,** 426-433. [204]

ROBACK, G.S., KRASNO, L.R. and IVY, A.F. Effect of analeptic drugs on the somnifacient effect of seconal and antihistaminics as measured by the flicker fusion threshold. *J. Appl. Physiol.* 1952, **4,** 566-744. [54, 67]

ROKEACH, M., ORAM, A., and MARR, J. N. The effects of meprobamate on analysis and synthesis in thinking. *J. Psychol.* 1959, **48,** 359-366. [213]

ROBERTS, J.M. Prognostic factors in the electro-shock treatment of depressive states. II. The application of specific tests. *J. Ment. Sci.* 1959, **105,** 703-713. [136]

ROSS, S., DARDANO, J. and HACKMAN, R.C. Conductance levels during vigilance task performance. *J. Appl. Psychol.* 1959, **43,** 65-69. [147]

ROTHBALLER, A.B. Studies on the adrenaline-sensitive component of the reticular activating system. *EEG Clin. Neurophysiol.* 1956, **8,** 603-621. [143, 372]

ROZHE, A., VORONIN, L.G., and SOKOLOV, Ye.M. Electroencephalographic research on temporary association with dampening of the orientation reflex in man. *Journal of Higher Nervous Activity imeni I. P. Pavlov,* 1958, **8,** 3-16. [357]

RUESCH, J. Dark adaption, negative after-images, tachistoscopic examinations and reaction time in head injuries. *J. Neurosurg.* 1944, **1,** 243-251. [203, 208]

RUETE *Lehrbruch der Ophthalmologie,* 1845, p. 75. [241]

SAGARA, M. and OYAMA, T. Experimental studies on figural after-effects in Japan. *Psychol. Bull.* 1957, **54,** 327-338. [256]

SAMUEL, I. Reticular mechanisms and behaviour. *Psychol. Bull.* 1959, **56,** 1-25. [143]

SANTISTEBAN, G. A. The response of the thymolymphotic system to graded doses of ethyl alcohol and its relationship to adrenocortical activity. *Quart. J. Stud. Alcohol*, 1961, **22**, 1-13. [330]

SARGANT, W. and SLATER, E. *An Introduction to Physical Methods of Treatment in Psychiatry*. London: Livingstone, 1956. [131, 372]

SAUCER, R.T. Processes of motion perception. *Science*, 1954, **120**, 806. [293]

SAUCER, R.T. and COPPINGER, M.W. Standard stimulus conditions for thresholds of apparent movement. *Amer. J. Psychol.* 1961, **73**, 435-439. [269]

SAUCER, R.T. and DEABLER, H.L. Perception of apparent motion in organics and schizoids. *J. Consult. Psychol.* 1956, **20**, 385-389. [269, 271]

SCHEIBEL, M.E. and SCHEIBEL, A.B. Structural substrates for integrative patterns in the brain stem reticular formation. In *Reticular Formation of the Brain*. London: Churchill, 1958. [358]

SCHMIDTKE, H. Über die Messung der psychischen Ermüdung mit Hilfe des Flimmertests. *Psychol. Forsch.* 1951, **23**, 409-463. [67]

SEAGER, C.P. Verification of the sedation threshold technique. *EEG Clin. Neurophysiol.* 1959, **11**, 606. [136, 137]

SEAGER, C.P. Problems in technique concerning the sedation threshold. *EEG Clin. Neurophysiol.* 1960, **12**, 910-913. [136, 137]

SHAGASS, C. The sedation threshold. A method for estimating tension in psychiatric patients. *EEG Clin. Neurophysiol.* 1954, **6**, 221-233. [133, 169, 171]

SHAGASS, C. A measurable neurophysiological factor of psychiatric significance. *EEG Clin. Neurophysiol.* 1957, **9**, 101-108.

SHAGASS, C. Neurophysiological studies of anxiety and depression. *A.P.A. Psychiat. Res. Rep.* 1958, **8**, 100 [169, 171]

SHAGASS, C., AZIMA, H. and SANGOWICZ, H. Effect of meprobamate in sustained high dosage on the electroencephalogram sedation threshold. *EEG Clin. Neurophysiol.* 1959, **11**, 275-283. [212]

SHAGASS, C. and JONES, A.L. A neurophysiological test for psychiatric diagnosis: results in 750 patients. *Amer. J. Psychiat.* 1958, **114**, 1002-1009. [133, 166, 167]

SHAGASS, C. and KERENYI, A.B. Neurophysiologic studies of personality. *J. Nerv. Ment. Dis.* 1958a, **126**, 141-147. [133]

SHAGASS, C. and KERENYI, A.B. The "sleep" threshold. A simple form of the sedation threshold for clinical use. *Canad. Psychiat. J.* 1958b, **1**, 101-109. [137, 169]

SHAGASS, C., MIHALIK, J. and JONES, A.L. Clinical psychiatric studies using the sedation threshold. *J. Psychosom. Res.* 1957, **2**, 45-55. [162, 171]

SHAGASS, C., MULLER, K. and ACOSTA, H.B. The Pentothal sleep threshold, as an indicator of affective change. *J. Psychosom. Res.* 1959, **3**, 253-270. [171, 174]

SHAGASS, C. and NAIMAN, J. The sedation threshold, manifest anxiety and some aspects of ego function. *A.M.A. Arch. Neurol. Psychiat.* 1955, **74**, 397-406. [133]

SHAGASS, C. and NAIMAN, J. The sedation threshold as an objective index of manifest anxiety in psychoneurotics. *J. Psychosom. Res.* 1956, **1,** 49-57. [19, 133, 170, 173]

SHAPIRO, M.B. A preliminary investigation of the effects of continuous stimulation in the perception of apparent movement. *Brit. J. Psychol.* 1954, **45**, 58 67. [237, 272, 293]

SHELDON, W.H. and STEVENS, S.S. The *Varieties of Temperament: a Psychology of Constitutional Difference.* New York: Harper Bros., 1942. [131]

SHERRINGTON, C. *The Integrative Action of the Nervous System.* Cambridge: Univ. Press, 1947. [253, 349]

SHIPLEY, W.G., KENNY, F.A. and KING, M.E. Beta apparent movement under binocular, monocular and interocular stimulation. *Amer. J. Psychol.* 1945, **58,** 545-549. [267, 268]

SIGAL, J.J., STAR, K.H. and FRANKS, C.M. Hysterics and dysthymics as criterion groups in the study of introversion-extraversion. *J. Abnorm. Soc. Psychol.* 1958, **57,** 143-148. [134, 135]

SIMONSON, E. and ENZER, N. Effect of Pervitin (desoxyephedrine) on fatigue of the central nervous system. *J. Industr. Hyg.* 1942, **24,** 205-209. [67]

SIMONSON, R., ENZER, N. and BLANKSTEIN, S.S. Effect of amphetamine (Benzedrine) on fatigue of the central nervous system. *War Med.* 1941, **1,** 690-695. [67]

SINGH, S.D. Conditioned emotional response in the rat: I. Consitutional and situational determinants. *J. Comp. Physiol. Psychol.* 1959, **52,** 574-578. [20]

SINGH, S.D. Conditioned emotional response in the rat: II. Effects of stimulant and depressant drugs. *J. Psychol. Res.* 1961. 5, 1-11.

SINGH, S.D. and EYSENCK, H.J. Conditioned emotional response in the rat: III. Drug antagonism. *J. Gen. Psychol.* 1960, **63,** 275-285. [20]

SINHA, S.N., FRANKS, C.M. and BROADHURST, P.L. The effect of a stimulant and a depressant drug on a measure of reactive inhibition. *J. Exp. Psychol.* 1958, **56,** 349-354. [19]

SKELTSOVA, N.I. *On the characteristics of some unconditioned reflexes to schizophrenic patients.* Trudy Vsesoyuzuri Nanchuro-Prakticheskoi Konferentzii, 1954. Moscow: Medgit, 1955. [4]

SLATER, P. A re-examination of some data collected by H.P. Hildebrand. In Eysenck, H.J. (Ed.), *Experiments in Personality.* London: Routledge and Kegan Paul, 1960. [166]

SMITH, G. A note on after-image serials in twins. *Acta Psychol.* 1957, **13,** 51-53. [69, 199, 204]

SMITH, K. The statistical theory of the figural after-effect. *Psychol. Rev.* 1952, **59,** 401-402.

SMITH, K.R. Visual apparent movement in the absence of neural interaction. *Amer. J. Psychol.* 1948, **61,** 73-78. [268]

SMITH, K.U. The neural centers concerned in the mediation of apparent movement vision. *J. Exp. Psychol.* 1940, **26,** 443-466. [269]

SMITH, K.U. and KAPPAUF, S.W. A neurological study of apparent movement. *J. Gen. Psychol.* 1940, **23,** 316-327. [269]

SMITH, S. and LEWTY, W. Perceptual isolation using a silent room. *Lancet*, 1959, **ii**, 342-345. [163]

SMITH, W.M. and GULICK, W.L. Visual contour and movement perception. *Science*, 1956, **124**, 316-317. [164]

SOKOLOV, E.N. Higher nervous activity and the problem of perception. *Vaprosy Psikhologii*, 1955, **1**, 58-66. and translated in Simon, B. (Ed.), *Psychology in the Soviet Union*. Routledge and Kegan Paul, 1957. [293]

SOLOMON, P. and WERNER, H. Studies on contour. III. Negative after-image. *Amer. J. Psychol.* 1952, **65**, 67-74. [205]

SOMNER, F.C. and WATTS, F.P. Rivalry between uniocular negative after-images and the vision of the other eye. *Amer. J. Psychol.* 1936, **48**, 109-116. [201]

SPEARMAN, C. *Abilities of man*. London: Macmillan, 1927. [7]

SPENCE, K.W. and TAYLOR, J.A. Anxiety and strength of the UCS as determinants of the amount of eyelid conditioning. *J. Exp. Psychol.* 1951, **42**, 183-188. [141]

SPERRY, R.W. and MINER, N. Pattern perception following insertion of mica plates into the visual cortex. *J. Comp. Physiol. Psychol.* 1955, **48**, 463-469. [293]

SPITZ, H.H. Neural satiation on spiral after-effect and similar movement after-effects. *Percept. Mot. Skills*, 1958, **81**, 207-213. [242, 243]

SPITZ, H.H. Some observations on the spiral after-effect. *Amer. J. Psychol.* 1959, **72**, 139-140. [235]

SPITZ, H.H. and BLACKMAN, L.S. A comparison of mental retardates and normals on visual figural after-effects and reversible figures. *J. Abnorm. Soc. Psychol.* 1959, **58**, 105-110. [235]

SPITZ, H.H. and LIPMAN, R.S. Some parameters in the perception of the spiral after-effect. *Percept. Mot. Skills*, 1959, **9**, 81. [235]

SPIVACK, G. and LEVINE, M. The spiral after-effect and reversible figures as measures of brain damage and memory. *J. Pers.* 1957, **25**, 767-778. [231, 236]

SPIVACK, G. and LEVINE, M. Personal communication, 1959. [240]

SPIVACK, G. and LEVINE, M. Spiral after-effect and measures of satiation in brain injured and normal subjects. *J. Pers.* 1959, **27**, 211-227. [233, 236, 238, 239, 250, 251]

STANDLEE, L.S. The Archimedes negative after-effect as an indication of memory impairment. *J. Consult. Psychol.* 1953, **17**, 317 [234]

STAUFFACHER, J.C. The effect of induced muscular tension upon various phases of the learning process. *J. Exp. Psychol.* 1937, **21**, 26-46. [142]

STAVRIANOS, B.K. The relation of shape perception to explicit judgements of inclination. *Arch. Psychol., N. Y.* 1945, **296**. [291]

STEINBERG, Hannah. Selective effects of an anaesthetic drug on cognitive behaviour. *Quart. J. Exp. Psychol.* 1954, **6**, 170-180. [176, 177]

STEINBERG, Hannah. Changes in time perception induced by an anaesthetic drug. *Brit. J. Psychol.* 1955, **47**, 273-279. [95]

STEINBERG, Hannah. 'Abnormal behaviour' induced by nitrous oxide. *Brit. J. Psychol.* 1956, **47**, 183-194. [95, 173]

STEINBERG, Hannah and SUMMERFIELD, A. Influence of a depressant drug on acquisition in rote learning. *Quart. J. Exp. Psychol.* 1957, **9,** 138-145. [192]

STERN, L.W. Die Wahrnehmung von Bewegungen Vermittelst des Auges. *Z. f. Psy. und Phy. de Sinnes,* 1894, **7,** 321-386. [229, 241]

STIGLER, M. Chronophorische Studien über den Umgebungskontrast. *Pflügers Arch. ges. Physiol.* 1910, **134,** 365-435. [27]

STILSON, D.W., GUNTHER, M.D. and GERTZ, B. Base rate and the Archimedes spiral illusion. *J. Consult. Psychol.* 1957, **21,** 435-437. [232]

STONE, G.C. Effects of some centrally acting drugs on learning of escape and avoidance habits. *J. Comp. Physiol. Psychol.* 1960, **53,** 1. [372]

STORMS, L.H. Discrepancies between factor analysis and multivariate discrimination among groups as applied to personality theory. *J. Ment. Sci.* 1958, **104,** 713-721. [166]

STORMS, L.H. and SIGAL, J.J. Eysenck's personality theory with special reference to the dynamics of anxiety and hysteria. *Brit. J. Med. Psychol.* 1958, **31,** 228-248. [236]

STROUD, J.B. The role of muscular tensions in stylus maze learning. *J. Exp. Psychol.* 1931, **14,** 606-631. [142]

SUMMERFIELD, A. and STEINBERG, H. Reducing interference in forgetting. *Quart. J. Exp. Psychol.* 1957, **9,** 146-154. [18]

SYLVESTER, J. Apparent movement and the Brown-Voth experiment. *Quart. J. Exp. Psychol.* 1960, **12,** 4. [294, 296, 300]

SZILY, V. Bewegungsnachbild und Bewegungs Kontrast. *Z. f. Psy. Phys. Sinnes,* 1905, **38,** 81-154. [229, 230]

SZILY, V. Zum Studium des Bewegungsnachbilds. *Z. f. Sinnesphys.* 1907, **42,** 109-114.

TAYLOR, J.A. The relationship of anxiety to the conditioned eyelid response *J. Exp. Psychol.* 1951, **41,** 81-92. [141]

TAYLOR, J.A. and SPENCE, K.W. The relationship of anxiety level to performance in serial learning. *J. Exp. Psychol.* 1952, **44,** 61-64. [142]

TEPLOV, B.M. *Typological Features of Higher Nervous Activity in Man.* (2 vols). Moscow: Acad. Pedagogical Sci., 1956, 1959. [1, 5]

TEUBER, A.L. and BENDER, M.B. Changes in visual perception of flicker apparent movement and real motion after cerebral trauma. *Amer. J. Psychol.* 1948, **3,** 246-247. [270]

THERON, P.A. Peripheral vasomotor reactions as indices of basic emotional tension and lability. *Psychosom. Med.* 1948, **10,** 335-346. [142, 143]

THOMPSON, M.E. A two-factor theory of inhibition. *Psychol. Rev.* 1960, **67,** 200-206. [4]

THOMPSON, S.P. Some new optical illusions. *Trans. Brit. Ass.* 1877, p. 32. [241]

THORPE, J.G. and BARKER, J.C. Objectivity of the sedation threshold. *Arch. Neurol. Psychiat.* 1957, **78,** 194-196. [136, 169, 173]

THOULESS, R.H. Phenomenal regression to the real object. *Brit. J. Psychol.* 1931, **21,** [289, 290]

TISSOT, R. and MOUNIER, M. On the dualism of the thalamic recruiting system (antagonism between the thalamic recruiting system and the ascending reticular system). *EEG Clin. Neurophysiol.* 1959, **11,** 675-680. [363]

TOCH, H.H. The perceptual elaboration of a stroboscopic presentation. *Amer. J. Psychol.* 1956, **69,** 345-358. [69]

TOCH, H.H. and ITTELSON, W.H. The role of past experience in apparent movement. *Brit. J. Psychol.* 1956, **47,** 195-207. [294]

TONG, J. E. Stress reactivity in relation to delinquent and psychopathic behaviour. *J. Ment. Sci.* 1959, **105,** 935-956. [166]

TONG, J.E. and MURPHY, I.C. A review of stress reactivity research in relation to psychopathology and psychopathic behaviour disorders. *J. Ment. Sci.* 1960, **106,** 1273-1295. [162]

TRAIN, L.E. Figural after-effects of apparent movement. *J. Gen. Psychol.* 1958, **59,** 157. [270]

TRASLER, G. *The Explanation of Criminality.* London: Routledge, Kegan Paul, 1962. [16]

TRAVIS, L.E. and HALL, M.E. Effect of visual after sensations upon brain potential patterning under different degrees of attention. *J. Exp. Psychol.* 1938, **22,** 472-479. [204]

TREADWELL, E. The effects of depressant drugs on vigilance and psychomotor performance. In Eysenck, H.J. (Ed.), *Experiments in Personality.* London: Routledge and Kegan Paul, 1960. [19]

TRENT, S.E. Narcosis and visual contour clarity. *J. Gen. Psychol.* 1947, **36,** 65-78. [63]

TROUTON, D. and EYSENCK, H.J. The effects of drugs on behavior. In Eysenck, H.J. (Ed.), *Handbook of Abnormal Psychology.* London: Pitman, 1960. [xi, 8, 18, 21, 24, 369, 373]

TROUTON, D.S. and EYSENCK, H.J. Psychological effects of drugs. In: Eysenck, H.J. (Ed.), *Handbook of Abnormal Psychology.* London: Pitman; New York: Basic Books, 1961.

TRUSS, C.V. and ALLEN, R.M. Duration of the spiral after-effect in cerebral palsy: an exploratory study. *Percept. Mot. Skills,* 1959, **9,** 216-218. [233, 236]

TSCHERMAK Zur Physiologie des Gesichtsorgans das Plateau-Oppelische Phaenomen und sein Platz in der Reiche der gleichartigen Erscheinungen *Militarmedizinisches Journal,* Juin-Juli, 1881. [241]

TUCKER, K. and WILLENSKY, H. A clinical evaluation of meprobamate therapy in a chronic schizophrenic population. *Amer. J. Psychiat.* 1957, **113,** 698-703. [212]

ULLMAN, A.D. The psychological mechanism of alcohol addiction. *Quart. J. Stud. Alcohol,* 1952, **13,** 602-608. [346]

ULLMAN, A.D. The first drinking experience of addictive and "normal" drinkers. *Quart. J. Stud. Alcohol,* 1953, **14,** 181-191. [346]

UNITED STATES EMPLOYMENT SERVICE. *General aptitude test battery.* (B-1001). Washington D.C., 1947. [176]

UTTAL, W.R. Inhibitory interaction of responses to electrical stimuli in the fingers. *J. Comp. Physiol Psychol.*1960, **52,** 47-51. [3, 83, 87, 107]

UTTAL, W.R. and COOK, Lovella On the absence of contralateral inhibitory interaction to electrical stimuli in the fingers. In press, 1961. [107]

VAN DER MERWE, A.B. The diagnostic value of peripheral vasomotor reactions in the psychoneuroses. *Psychosom. Med.* 1948, **10,** 347-354. [142, 144, 160]

VAUGHN, J. and DISERENS, C.M. The relative of effects various intensities of punishment on learning and efficiency. *J. Comp. Psychol.* 1930, **10,** 55-66. [142]

VENABLES, P.H. Factors in the motor behaviour of functional psychotics. *J. Abnorm. Soc. Psychol.* 1959, **58,** 153-156. [139]

VENABLES, P.H. and TIZARD, J. The effect of stimulus light intensity on reaction time of schizophrenics. *Brit. J. Psychol.* 1956, **47,** 144-146. [252]

VENABLES, P.H. and TIZARD, J. Paradoxical effects in the reaction time of schizophrenics. *J. Abnorm. Soc. Psychol.* 1956, **53,** 220-224. [252]

VOGEL, Muriel. Low blood alcohol concentrations and psychological adjustment as factors in psychomotor performance. *Quart. J. Stud. Alcohol,* 1958, **19,** 573-589. [330, 347]

VOGEL, M.D. The relation of personality factors to GSR conditioning of alcoholics: an exploratory study. *Canad. J. Psychol.* 1960, **14,** 275-280. [330, 347]

VOGEL, M.D. GSR conditioning and personality factor in alcoholics and normals. *J. Abnorm. Sc. Psychol.* 1961, **63,** 417-421. [14]

VON BONIN, G., GAROL, H.W. and MCCULLOCH, W.S. The functional organisation of the occipital lobe. *Biol. Symposium,* 1942, **7,** 165-192. [293]

WALLS, G.L. Interocular transfer of after-images. *Amer. J. Optom.* 1953, **30,** 57-64. [229]

WALSH, E.G. *Physiology of the Nervous System.* London: Longmans, Green, 1957. [356, 358, 361, 363]

WALTHALL, W.J. Field strength of the Kohler effect. *J. Genet. Psychol.* 1949, **41,** 27-32. [293]

WALTON, D. Drug addiction and habit formation — an attempted integration. *J. Ment. Sci.* 1960, **106,** 1195-1229. [166]

WALTON, H.V. A study of retinal correspondence by after-image methods. *Amer. J. Optom.* 1952, **29,** 90-103. [202]

WARD, A.A. Efferent functions of the reticular formation. In *Reticular Formation of the Brain.* London: Churchill, 1958. [363]

WEBB, W.B. A motivational theory of emotions. *Psychol. Rev.* 1948, **55,** 329-335. [142]

WEIL-MALHERBE, H. The concentration of adrenaline in human plasma and its relation to mental activity. *J. Ment. Sci.* 1955, **101,** 733-755. [144, 167]

WEINSTEIN, E.A. and MALITZ, S. Changes in symbolic expression with sodium amytal. *Amer. J. Psychiat.* 1954, **111,** 198-206. [232]

WEISKRANTZ, L. Figural after-effects in stroboscopic motion. *Quart. J. Exp. Psychol.* 1950, **2,** 113-118. [272]

WEISKRANTZ, L. An unusual case of after-imagery following fixation of an "imaginary" visual pattern. *Quart. J. Exp. Psychol.* 1950, **2,** 170-175. [270]

WENGER, M.A. The measurement of individual differences in autonomic balance. *Psychosom. Med.* 1941, **3,** 427-434. [142]

WENGER, M.A. Studies of autonomic balance in Army Air Forces personnel. *Comp. Psychol. Monogr.* 1948, **19,** 1-111. [142]

WERNER, H. Studies in contour. I. Qualitative analyses. *Amer. J. Psychol.* 1935, **47,** 40-64. [70, 71, 72, 75, 91, 119]

WERNER, H. Studies in contour: strobostereoscopic phenomena. *Amer. J. Psychol.* 1940, **53,** 418-422. [69, 72, 75, 120]

WERNER, H. and THUMA, A. A deficiency in the perception of apparent movement in children with brain injury. *Amer. J. Psychol.* 1942, **55,** 58-67. [269, 285]

WERTHEIMER, M. Experimentelle Studien über das Sehen von Bewegung. *Z. Psychol.* 1912, **61,** 161-265. [292, 296]

WERTHEIMER, M. The differential satiability of schizophrenic and normal subjects: a test of deduction from the theory of figural after-effects. *J. Gen. Psychol.* 1954, **51,** 291-299. [258]

WERTHEIMER, M. Figural after-effect as a measure of metabolic efficiency. *J. Pers.* 1955, **24,** 56-73. [258]

WERTHEIMER, M., LEVINE, H. and WERTHEIMER, N. The effect of experimentally induced changes in metabolism on perceptual measures of metabolic efficiency. *Percept. Mot. Skills,* 1955, **5,** 173-176. [258].

WERTHEIMER, M. and WERTHEIMER, N. A metabolic interpretation of individual differences in figural after-effects. *Psychol. Rev.* 1954, **61,** 279-280. [258]

WEST, G. B., SHEPHERD, D.M. and HUNTER, R.B. Adrenaline and noradrenaline concentrations in adrenal glands at different ages in some diseases. *Lancet,* 1951, **261,** 966-969. [167]

WEVER, E.G. Figure and ground in the visual perception of form. *Amer. J. Psychol.* 1927, **38,** 194-226. [69, 70, 72]

WILLETT, R.A. The effects of depressant drugs on learning and conditioning In Eysenck, H.J. (Ed.), *Experiments in Personality.* London: Routledge and Kegan Paul, 1960. [19]

WILLETT, R.A. The effects of psychosurgical procedures on behaviour. In Eysenck, H.J. (Ed.), *Handbook of Abnormal Psychology.* London: Pitman, 1960; New York: Basic Books, 1960. [7, 321]

WILLIAMS, H.L., LUBIN, A. and GOODNON, J.J. Impaired performance with acute sleep loss. *Psychol. Monogr.* 1959, **73,** No. 14. [147]

WILSON, A. and SCHILD, H.O. *Applied Pharmacology,* 9th edition, London: Churchill, 1959. [175, 354, 368, 373, 374, 375, 376]

WINTERSTEIN, H. The actions of substances introduced into the cerebrospinal fluid and the problem of intercranial chemoreceptors. *Pharmacol. Rev.* 1961, **13,** 71-107. [330]

WITHERS, W.C.R. A new tachistoscope and cycling timer. *Brit. J. Psychol.* 1954, **45,** 217-219.
WITKIN, H.A. *et. al. Personality through Perception.* New York: Harper, 1954. [293]
WOHLGEMUTH, A. On the after-effect of seen movement. *Brit. J. Psychol. Monogr. Suppl.* **1**, 1911. [237, 240, 241, 242]
WOODWORTH, R.S. *Experimental Psychology.* London: Methuen, 1950. [209]
WUNDT, W. *Grundzüge der physiologischen Psychologie.* Leipzig: Engelmann, 1874. [9, 241]

YATES, A.J. Abnormalities of psycho-motor functions. In Eysenck, H.J. (Ed.), *Handbook of Abnormal Psychology.* London: Pitman, 1960. [142]
YERKES, R.M. and DODSON, J.D. The relation of strength of stimulus to rapidity of habit-formation. *J. Comp. Neurol. Psychol.* 1908, **18,** 459-482. [142]
YOUNG, P.T. *Motivation of Behavior.* New York: Wiley, 1936. [9]
YOUNG, P.T. *Motivation and Emotion.* New York: Wiley, 1961. [9]

ZEHFUSS, G. Über Bewegungsnachbilder. *Wiedemann's Annalen d. Physik und Chemie*, 1880, **9,** 672-676. [241]
ZOLLNER, F. Über eine neue Art von Pseudoskopie und ihre Beziehung zu der von Plateau und Oppel beschriebenen. *Pogg. Ann.* 1860, **110,** 500-523. [241]

SUBJECT INDEX